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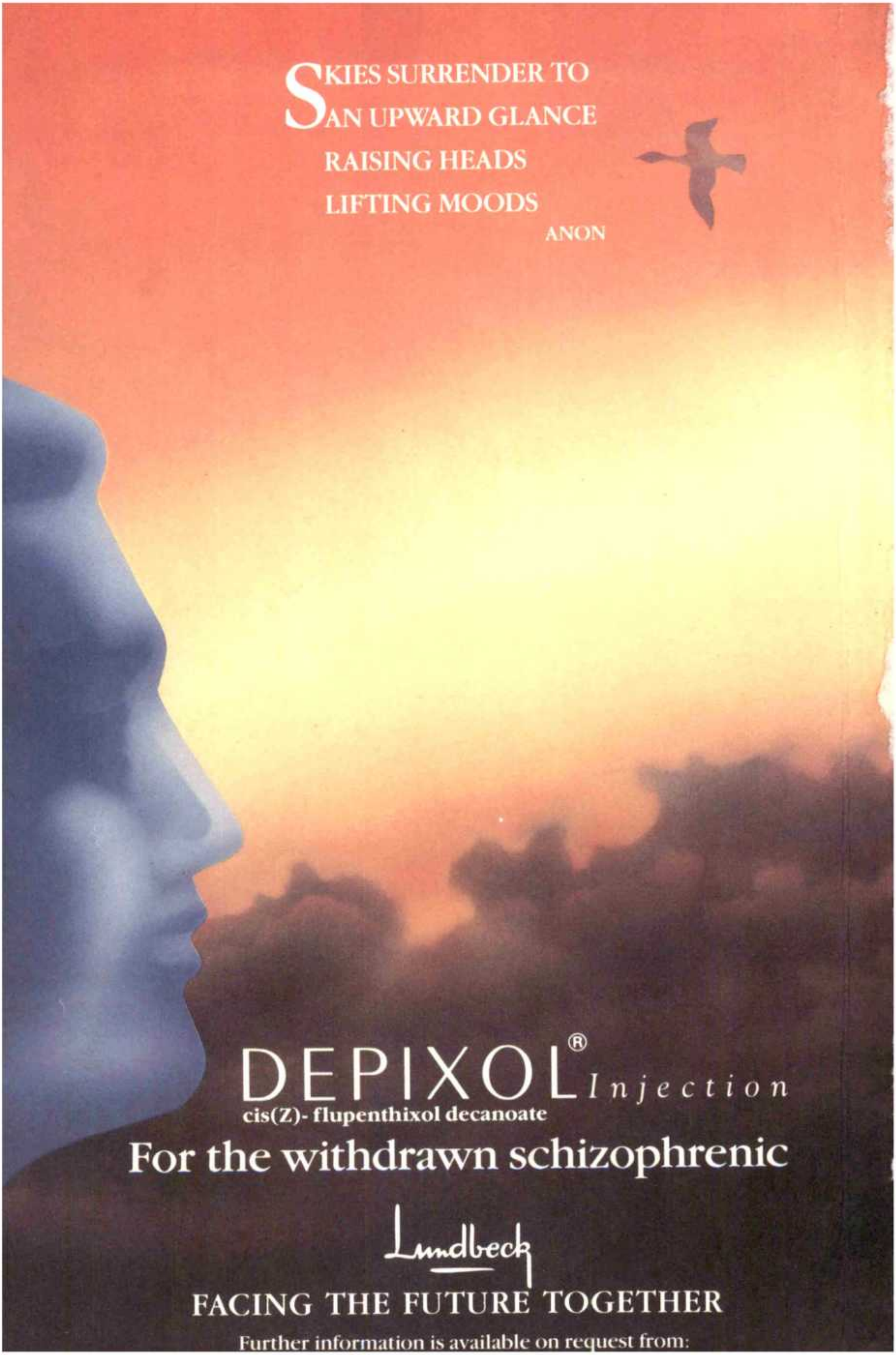

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Annotation

Confusions Concerning Sleep Disorders and the Epilepsies in Children and Adolescents

GREGORY STORES

There are interesting parallels between sleep disorders and the epilepsies. Firstly, both terms refer to a group of conditions differing in cause, clinical manifestation, natural history and impact on the lives of patients and their relatives. Secondly, the investigation and management of both sets of conditions require a combined physical and psychological approach. Thirdly, neither field is usually covered systematically (if at all) in medical education, although some initiative has been taken in the USA for the introduction of these areas into the curriculum of medical schools (Medical School Curriculum Task Force, 1988). This relative neglect no doubt explains the fourth parallel – that in both sleep disorders and the epilepsies misdiagnosis or inadequate diagnosis appears to be common.

This account is concerned principally with these diagnostic problems, of which there are different varieties. The types of diagnostic error and the conditions in which diagnostic confusion may well occur are set out below:

(a) sleep disorders confused with each other:
  nightmares
  night terrors and other 'arousal disorders'
  rapid eye movement (REM) sleep behaviour disorder
(b) sleep disorders misdiagnosed as epilepsy:
  arousal disorders
  REM sleep behaviour disorder
  head-banging and related phenomena
  nocturnal enuresis
  obstructive sleep apnoea
  automatic behaviour disorder
  idiopathic central nervous system (CNS) hypersomnia with sleep drunkenness
(c) epilepsy misdiagnosed as sleep disorder:
  complex partial seizures of temporal lobe origin
  complex partial seizures of frontal lobe origin
  nocturnal/hypnogenic paroxysmal dystonia
  episodic nocturnal wanderings
  non-convulsive status epilepticus

It is suggested that if the salient features of these conditions are known they will usually be recognised, provided that a detailed account is obtained of each patient's episodes of disturbed behaviour, the timing of the episodes, and the circumstances in which they occur. In some cases, video and physiological monitoring will be needed. Although emphasis is placed on children and adolescents, many of the conditions and issues are relevant to adult psychiatry. Firstly, some comments on present classification schemes are required.

Classification of sleep disorders

Textbooks of paediatrics and child psychiatry often understate the range of sleep disorders. There may simply be brief accounts of night wakening, nightmares, sleep walking and night terrors. The true diversity of sleep disorders is demonstrated in the classification of sleep and arousal disorders compiled by the Association of Sleep Disorders Centers (1979) which however needs some modification to include those sleep disorders and types of sleep behaviour seen specifically in infants and children (Ferber, 1985). At any age, four main categories of sleep problems can be identified: disorders of initiating and maintaining sleep (insomnias), disorders of excessive daytime sleepiness (hypersomnias), disorders of the sleep–wake cycle, and episodic types of behaviour made worse by sleep or occurring exclusively during sleep (parasomnias). Most confusions concerning sleep disorders and epilepsies seem to consist of difficulty in distinguishing between nocturnal epileptic attacks and other parasomnias.

Perhaps especially in children, there is a need to distinguish between sleep disorders and sleep behaviour. The former term implies a problem to the child or parents, whereas a sleep behaviour may or may not be a problem; it may simply reflect a certain phase of development. For example, it is usual for infants to wake repeatedly during the night (Anders, 1979). Such wakenings are only a problem if the child habitually cannot settle back to sleep and cries or disturbs the parents in other ways. Other types of sleep behaviour which are not necessarily problematic include teeth grinding, head-banging or rocking, and snoring, although each of these can be a pointer to a serious condition.

Classification of epileptic seizures and syndromes

A scheme for classifying epileptic attacks (seizures) has been promoted internationally for the last 20 years (Gastaut, 1970; Commission on Classification and Terminology of the International League Against Epilepsy, 1981). Whatever the misgivings expressed by traditionalists, the scheme has the undoubted advantage of encouraging careful clinical observation of seizures in place of simplistic categorisation in terms such as '*grand mal*' and '*petit mal*'. It has become clear that what used to be called 'temporal lobe attacks', for example, are in fact a miscellaneous group of seizures and that the term is of limited clinical value.

Attempts have also been made to solve the more difficult problem of classifying the epilepsies or epileptic syndromes. An account by the Commission on Classification and Terminology of the International League Against Epilepsy (1989) demonstrates the need for accurate investigation of the clinical, electrographic (EEG), aetiological, developmental and other features of the individual case. This degree of assessment permits accurate classification in the vast majority of children (Viani *et al*, 1988). The detailed evaluation required for accurate classification of a patient's seizures and syndrome is not merely an academic exercise: it is of fundamental importance in deciding what investigations are required, the choice of treatment, and in estimating prognosis. Benign centrotemporal (Rolandic) epilepsy of childhood (Blom & Heijbel, 1982) illustrates this point. This is a very common form of childhood epilepsy, probably often grouped previously with other seizure disorders under the general term of 'temporal lobe epilepsy', but now known to be unlike most other partial epilepsies in very important ways. If its characteristic clinical and EEG features are identified, the fear of a structural brain abnormality can be removed and all those concerned can be reassured that spontaneous remission is almost certain by puberty. Myoclonic seizures are another case in point, forming part of quite different syndromes. They may be part of a benign and transient disorder, or a feature of progressive neurodegenerative disease.

Sleep disorders confused with each other

There is a tendency to confuse different types of acutely disturbed behaviour in sleep and to call any such disturbance a 'nightmare'. Sometimes textbooks expressly equate nightmares with night terrors or one is said to be a variety of the other. In fact, fundamental differences exist between nightmares and night terrors (and other so-called 'arousal disorders') and in recent times accounts of yet other types of sleep disorder characterised by dramatic behaviour have been published.

Nightmares are frightening dreams occurring in REM sleep during the later part of the night when that stage of sleep is most abundant. In a nightmare the child will wake and give an account of a frightening dream experience. The term 'arousal disorder' (Broughton, 1968) covers a spectrum of behaviour types which have in common that they are sudden partial wakenings towards the end of a period of deep (stages 3 and 4) non-REM sleep, at the point of transition from this stage to a lighter level of sleep. As deep non-REM sleep occurs mainly in the first part of sleep, primarily in the first sleep cycle, these disorders happen within the first three hours or so after the child falls asleep. During the episode the child remains deeply asleep, and therefore inaccessible, but also exhibits behaviour more reminiscent of the awake state. Afterwards there is little or no recall of the events.

There is a spectrum of clinical accompaniments to these arousals, from mild changes to very dramatic behaviour, and the nature of the episode varies somewhat with the age of the child. There may be no more than turning over or perhaps brief moaning; or motor activity and vocalisations may be more marked, with crying and thrashing about in the young child or, at an older age, sitting up, staring, shuffling about in bed, with incoherent muttering or more formed sentences ('sleep talking') and apparent confusion. Alternatively, the child may get out of bed and walk about in a semi-purposeful way ('sleep walking') and indulge in conversations. There may be signs of agitation sometimes amounting to a state of terror, with crying out, intense autonomic overactivity (much greater than in nightmares) and avoidance behaviour ('night or sleep terrors'). Perhaps older children and adolescents in particular may rush about in a terrified state or even dash out of the house. Guilleminault & Silvestri (1982) provide some striking examples of these types of disturbed behaviour in adolescents (and adults) during non-epileptic parasomnias.

Therefore there are important differences between nightmares and arousal disorders regarding timing, clinical features (including the child's awareness and recall) and basic physiology (Kales *et al*, 1987). The form of management required is also different in these two types of condition. In general it is appropriate and possible to comfort a child who has woken from a nightmare. Attempts to wake a child in a night terror or agitated sleep-walking episode may cause him/her confusion and distress; calm reassurance when the acute episode is over and making the environment safe to prevent accidental injury are more useful measures.

In recent years another type of sometimes dramatic behaviour disorder during sleep has been described. This is the 'REM sleep behaviour disorder' (Mahowald & Schenck, 1989), in which the person is able to act out his/her dreams. This is possible because of a disturbance of REM sleep mechanisms, whereby skeletal muscular tone is preserved during REM sleep instead of being reduced as is normally the case. If the content of the dream is violent patients may injure themselves or others, perhaps seriously. Clonazepam is said to be an effective treatment. At present it seems that this particular sleep disorder is likely to be encountered in children only very rarely. Most reports have been concerned with middle-aged or elderly males, but there have been some reports (Barros-Ferreira *et al*, 1975; Schenck *et al*, 1986; Herman *et al*, 1989) suggesting the same condition in children between the ages of two years (this child was initially thought to have epilepsy) and ten years. These children usually show signs of a brain-stem lesion, presumably affecting the brain-stem nuclei which control REM sleep. Many of the adult patients in whom this disorder has been described had previously been diagnosed as having nightmares (Mahowald & Schenck, 1989).

Sleep disorders misdiagnosed as epilepsy

There seems to have been a tendency, perhaps on the part of psychiatrists in particular, to consider epilepsy a likely explanation of apparently unprovoked episodes of violent or otherwise antisocial behaviour. Several reviews (e.g. Treiman & Delgado-Escueta, 1983) have demonstrated that this type of behaviour in the daytime is very unlikely to be epileptic in origin. The same appears to be true of such behaviour during the night. Other conditions, especially arousal disorders, are a more likely explanation and must be distinguished from epilepsy in spite of sometimes sharing such features as abrupt onset, confusion, amnesia and injury.

There have been various reports suggesting that violence, including homicidal behaviour, can occur as part of a sleep disorder and, in some, consideration of the underlying cause has mistakenly emphasised the possibility of epilepsy. Oswald & Evans (1985) described three cases, including that of a 14-year-old boy, where serious violence had been perpetrated on a sleeping partner or relative during what was eventually considered to be sleep walking. These authors reviewed other reports of apparently similar cases and referred to the outmoded notion that what are now called arousal disorders are a form of epilepsy. Scott (1988) described another such case in which the diagnosis of epilepsy was mistakenly entertained repeatedly and the patient advised not to pursue his occupation as an ambulance driver. As pointed out by Morehouse (1989), the REM sleep behaviour disorder should also be considered as an alternative diagnosis in cases exhibiting violence during sleep. In a series of 11 carefully investigated cases of violence in sleep, the youngest being 14 years old, seven were diagnosed as arousal disorder and the rest as single cases of REM sleep behaviour disorder, sleep apnoea, idiopathic CNS hypersomnia, and alcohol-related sleep disorder. Epilepsy did not feature at all (Broughton, personal communication).

Behaviour during sleep does not have to be dramatic for epilepsy to be considered. In the epilepsy and sleep disorders clinics at the Park Hospital for Children in Oxford, lesser forms of arousal disorder and even head rolling have been encountered in which epilepsy had been diagnosed or considered likely.

Case 1

JL, a boy with the Pierre Robin syndrome, was eight years old when he began to have night-time episodes lasting between 10 seconds and 10 minutes of shouting, incoherent speech, vigorous random leg movements and a frightened facial expression. These episodes occurred two to three times a month. Less frequently he would walk downstairs from his bedroom and carry out repetitive acts, such as walking around the table, in an unresponsive state. At the age of 11, at a time when his behaviour was generally disturbed, he was said to have had episodes during the day of diminished responsiveness and facial grimacing lasting up to three hours. An EEG was reported as "consistent with multifocal cortical epilepsy" and antiepileptic drugs were introduced. JL's daytime episodes subsided over the next 12 months but he was referred to the Park Hospital epilepsy service at the age of 13 because of persistence of his night-time attacks. Overnight video, sleep studies and oximetry showed repeated nocturnal episodes arising from stage 4 non-REM sleep with no evidence of seizure discharge or sleep apnoea. Review of the original EEG showed that it contained no convincing abnormality. The night-time episodes were diagnosed as an arousal disorder. On further inquiry the earlier day-time episodes seemed likely to have been a behaviour problem. Antiepileptic drugs were withdrawn and the need for psychological help was emphasised. Since then JL's attacks have become progressively less frequent.

Case 2

SO was said to have always been a light sleeper and to have taken a long time to get to sleep. As a young child she would chatter to herself until 1.00 a.m. or roll from side to side for long periods, sometimes banging her head on the pillow. This behaviour was still evident by the age of three and gradually worsened over the next few years. The rocking was sometimes accompanied by growling and occurred repeatedly during the night as well as occasionally during the day, associated with hand flapping. SO was sleepy during the day and both her learning and her general behaviour were a problem, particularly at school. She had had a tonic–clonic seizure associated with fever at the age of two years, which was said to have been followed by a right Todd's paresis, and at about the same age she underwent bilateral reimplantation of her ureters because of congenital renal abnormalities. When she was seven, SO was referred for EEG to explore the possibility that her persistent night-time attacks were epileptic in nature. Combined video and cassette EEG recordings overnight showed that the rolling episodes occurred as SO woke repeatedly through the night. Each episode lasted about 10 seconds and occurred in quick succession for a total duration of between 10 and 30 minutes until she settled back to sleep again. No convincing seizure discharge was seen at any stage. Sleep structure was disrupted, with short overall sleep time. It was considered that SO was suffering from unusually persistent head rocking and banging associated with disturbed daytime behaviour and that her disrupted sleep was the cause of the daytime sleepiness.

There are other more basic sleep phenomena that can raise the possibility of nocturnal seizures. An obvious example is enuresis, which is common and not usually of epileptic origin. Nevertheless, this possibility needs to be considered if there is any suggestion of convulsions during the night or if there is no response to conventional treatment for enuresis. Enuresis is one of several possible manifestations of obstructive sleep apnoea in children, which tend to be more varied than in adults where loud snoring is the predominant sign. Other nocturnal symptoms in this condition that might be construed as evidence of seizure activity are difficulty in breathing, restless sleep and intense sweating (*Lancet*, 1989).

There are two other sleep disorders which, although apparently uncommon, have clinical features which could be confused with complex partial seizures of temporal lobe origin or non-convulsive status epilepticus. Alternatively, they may be misconstrued as a primarily psychiatric disorder. The first is the automatic behaviour syndrome (Guilleminault *et al*, 1975*a*). This is said to be characterised by a continuation of activities in an altered state of consciousness, sometimes with hallucinations, followed by complete amnesia. Each episode lasts a few seconds to several hours, during which time repetitive 'microsleeps' occur. During an episode the patient appears blank and out of touch with his/her surroundings. There may be drooping or fluttering of the eyelids and vocalisations of varying length and meaning. The attacks tend to occur in circumstances that predispose to drowsiness. Sense of time is lost and the patient's activities can be a source of embarrassment, perplexity or danger. This condition has been associated mainly with the narcolepsy syndrome but also with other causes of daytime sleepiness, including sleep apnoea. Reported series include children (Guilleminault *et al*, 1975*b*). Some of the ways in which automatic behaviour of this type is different from temporal lobe complex partial seizures (of usual duration) are absence of aura, wider range of duration, alerting effect of stimulation and lack of both affective change and post-ictal symptoms (Pedley, 1983). However, the distinction between the automatic behaviour syndrome and non-convulsive status epilepticus, whether absence status or complex partial status (Stores, 1986), may be difficult from the clinical features alone, but physiological recording during the episodes would demonstrate microsleeps in the former condition and seizure discharge in the latter.

The same type of distinction might need to be made between 'idiopathic CNS hypersomnia with sleep drunkenness' (Roth *et al*, 1972) and non-convulsive status epilepticus. The age of onset in idiopathic CNS hypersomnia is said to be from 15 years onwards. Salient features are great difficulty getting up in the morning, in spite of an apparently sound night's sleep, and inability to become fully alert for a long time after waking. Considerable difficulties arise at school, at work, or elsewhere, because of extreme tiredness, disorientation, incoordination and often automatic behaviour, as previously described. Unfortunately the prospects for improvement are poor.

Epilepsy misdiagnosed as sleep disorder

The opposite form of misdiagnosis is failure to recognise nocturnal epileptic attacks as epilepsy at all. This is particularly liable to happen in the case of non-convulsive seizures, which form a high proportion of seizure types.

Interictal focal epileptic discharges and partial seizures are provoked by both REM and non-REM sleep to the extent that some patients have exclusively nocturnal attacks. Seizures of a complex partial type are those most likely to be confused with non-epileptic parasomnias, especially arousal disorders, because their clinical features, especially automatic behaviour, overlap. Most complex partial seizures arise from a temporal source, therefore it is this form of seizure that numerically is most likely to be mistaken for a non-epileptic parasomnia. However, perhaps as many as 30% of complex partial seizures have an extra-temporal origin and of these possibly a third arise from a seizure source in the frontal area, mainly orbito-frontal or mesial-frontal (Williamson & Spencer, 1986).

Complex partial seizures of frontal origin have only relatively recently been described (especially in children), with the result that they are generally unknown to clinicians. Mainly because of their sometimes bizarre nature, including complicated automatisms and vocalisations, those occurring in the daytime are often construed as 'hysterical' or as evidence of some other form of psychiatric disorder. This appears to be true for both adults and children. When attacks of this sort occur in children at night (which many do), they may well be diagnosed as 'nightmares' (Stores & Zaiwalla, 1989). This mistake is difficult to justify, not only because of the nature of the automatisms in these attacks and the lack of any evidence of dreaming, but also because of their very frequent occurrence (perhaps many each night) and their abrupt onset and termination, sometimes after only a matter of several seconds. Conventional EEG recordings can be of limited value in this form of epilepsy, although there is some suggestion that ictal recordings which emphasise frontal and prefrontal electrode placements may provide useful evidence of a frontal seizure source (Stores & Zaiwalla, 1989).

Case 3

From the age of three years CJ had episodes of inappropriate behaviour during the day which were initially diagnosed as hysteria. The diagnosis was changed to epilepsy when she started to report abdominal sensations at the start of some of these episodes. Antiepileptic drug treatment was associated with a reduction of the daytime episodes but at the age of nine years CJ began to have frequent night-time attacks consisting of thrashing movements of her limbs, grunting, sitting up and rocking from side to side for up to about 30 seconds. When no definite EEG changes were recorded during two of these night-time episodes the diagnosis was revised again to 'pseudoseizures' and psychiatric treatment was provided, including the use of amitriptyline. The attacks increased in frequency. Reassessment at the Park Hospital included overnight video and cassette EEG monitoring during many of these attacks which occurred throughout the night. The episodes were brief and mainly consisted of complex motor automatism including repeated flexion–extension or scissor movements of the legs. At the onset of these attacks a left pre-frontal sharp-wave discharge was seen repeatedly before more widespread EEG changes occurred. The attacks were diagnosed as complex partial seizures of left frontal origin. No evidence of a structural abnormality was found.

Crowell & Anders (1985) described a ten-year-old child diagnosed as having 'hypnogenic paroxysmal dystonia' which they felt should be included in the differential diagnosis of epileptic attacks and pseudoseizures. The originators of this term (Lugaresi *et al*, 1986) have more recently preferred the term 'nocturnal paroxysmal dystonia' and have documented two forms: short and long-lasting. Attacks in the shorter form have many features in common with complex partial seizures of frontal origin and may well be further examples of that form of epilepsy (Tinuper *et al*, 1990); the nature of the longer episodes remains obscure.

Another condition characterised by disturbed behaviour during the night and possibly epileptic in nature is 'episodic nocturnal wanderings' as described originally by Pedley & Guilleminault (1977) and more recently by Maselli *et al* (1988). Onset is usually in the late teens. Episodes consist of frequent stereotyped attacks of screaming, walking about and complex automatisms, including violent behaviour, during sleep. These episodes are more complicated, violent and longer lasting (up to several minutes) than frontal complex partial seizures or nocturnal paroxysmal dystonia. Their variable time of occurrence during the night does not particularly suggest an arousal disorder from deep non-REM sleep. Some of those with this condition were said to have 'epileptiform' activity in their inter-attack EEGs, but seizure discharge was not recorded at the time of their attacks. A good response to antiepileptic drugs (mainly carbamazepine) was usually obtained. Whether this condition is distinct from the epileptic and non-epileptic disorders discussed earlier remains uncertain. Oswald (1989) comes down firmly on the side of it being night terrors with sleep walking.

Finally, as suggested earlier, the various types of non-convulsive status epilepticus can be confused with sleep disorder. The clinical manifestations of this form of epilepsy are varied. Textbook descriptions emphasise the more dramatic forms in which the patient (adult or child) is stuporose or 'pseudodemented', with clinical features somewhat similar to the long daytime episodes of sleep disorder described previously, sometimes including ataxia. But this obvious impairment of performance and behaviour is only one end of a spectrum. At the other extreme the clinical accompaniments of even gross continuous EEG abnormalities can be very subtle (Stores, 1986). Towards this less obvious extreme a salient feature can be that of a mild degree of sleepiness as illustrated by some of a series of children with non-convulsive status epilepticus seen at the Park Hospital for Children.

Case 4

From the age of seven JH began to fall asleep repeatedly during the day in spite of apparently sleeping well at night. At various times every day he went to sleep and was confused when he eventually awoke about three to four hours later. On other occasions he would simply yawn, stretch and complain of feeling tired. JH had a past history of a febrile convulsion at six months of age and infrequent tonic–clonic seizures since he was 18 months old.

Medication at the onset of his daytime sleepiness was carbamazepine and sodium valproate in modest dosage. Investigation at the Park Hospital, including cassette monitoring and combined EEG/video recordings, showed only occasional brief bursts of generalised polyspike and wave activity when JH was alert, but continuous generalised spike wave discharge during his tired and sleepy periods. These were diagnosed as episodes of generalised non-convulsive status epilepticus. Withdrawal of carbamazepine and an increase in the dosage of sodium valproate abolished the episodes completely.

Coexistence of epilepsy and sleep disorder

Night-time episodes of disturbed behaviour need not be exclusively either sleep disorder or epilepsy. Epilepsy is common – its prevalence rate in children has been estimated to be at least 1% (Rose *et al*, 1973). Sleep disorders are much more common than epilepsy (Simonds & Parraga, 1982). Epilepsy and sleep disorders therefore may well coexist in some children. As children with epilepsy have high rates of emotional disturbance (Stores, 1981), sleep problems associated with emotional upset are likely to be over-represented in this group. In fact, information on sleep problems in children with epilepsy is very limited, but the findings of Zaiwalla (1989) suggested a generally higher rate of sleep problems in such children compared with controls, and Tassinari *et al* (1972) have described night terrors in children with epilepsy.

Clinical assessment and physiological monitoring in sleep disorders

It is generally agreed that the basis of accurate diagnosis of episodic disturbances of behaviour is careful description of the subjective and objective nature of the attacks (including the precise sequence of events), the circumstances in which they occur, as well as their timing. The range of possible diagnoses, and the characteristic features of each condition, must be known.

However, detailed descriptions can be difficult to achieve, certainly in the case of night-time attacks in children. Encouraging parents to describe their observation in as systematic and as detailed a fashion as possible (perhaps with the aid of a diary) can improve the quality of reporting, and further information from siblings can be very helpful, especially if they share a bedroom with the child in question. However, parents or even siblings are unlikely to witness the onset of attacks (of particular importance in the case of seizures) and may only report the more dramatic features. As a result cases are encountered where a confident clinical diagnosis cannot be made because of inadequate information. It is in such a situation that the risk of a mistaken diagnosis such as 'nightmares' or 'epilepsy' is greatest.

In these circumstances overnight video recordings can be very instructive about the nature of the attacks and their timing. Discrepancies may well be observed between the account given by relatives (or even hospital night staff) and the features revealed by analysis of the video tape. At present this facility is very little used but there is no reason why it should not be employed more widely, including recordings at home with parents' own equipment or a video system loaned by the hospital's clinical neurophysiology department. If this is not feasible then such recordings should usually be possible in any child psychiatry or paediatric in-patient unit, residential school or other setting where the child sleeps. Diagnostic accuracy can be further increased by simultaneously recording the EEG and, if necessary, other sleep parameters for conventional sleep analysis (Rechtschaffen & Kales, 1968). As illustrated in the above case reports, EEG and sleep recordings can now be conveniently achieved at home, or wherever else the child is sleeping, by means of a portable cassette system (Stores & Bergel, 1989).

Conclusions

Nocturnal disturbances of behaviour are common, especially in children. The various conditions in which such behaviour can occur seem to be a source of diagnostic confusion, perhaps especially when the possibility of epilepsy arises. The classification schemes now available for both sleep disorders and the epilepsies, as well as recent accounts of previously unrecognised disorders of both types, can serve as a useful aid to accurate clinical diagnosis.

Where diagnostic difficulty remains, especially because of inadequate clinical information, video and physiological monitoring can be helpful. Such investigations are now possible, even as out-patient procedures, by means of recently developed recording systems. These facilities are not often provided by departments of clinical neurophysiology at the present time, not because highly specialised expertise is required, but principally because of limited demand from clinicians. An all-round knowledge of the different varieties of night-time disturbance of behaviour is needed, combined with an increased awareness of the ways in which accurate diagnoses can be made.

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Review Article

Schizophrenia: A Neuropathological Perspective

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It is probable that all schizophrenics have abnormalities in the medial temporal lobe, which differ in degree but not in kind. The structures of the medial temporal lobe are believed to have a crucial role in the integration and processing of the output from the association cortex. Dysfunction of this system could result in the clinical symptoms that form the core of the schizophrenia syndrome. The structural differences appear to fit the profile of a disturbance in the normal pattern of brain development. The asymmetrical patterns of normal brain development explain how such a disturbance simultaneously affecting both hemispheres could, disproportionately, affect the left (dominant) hemisphere. Epidemiological and pathological evidence points to aberrant genetic mechanisms as being the cause of the developmental anomaly in the majority of cases; environmental factors probably play a minor role. Despite the great progress made in solving the enigma of the structural changes in the brains of schizophrenics, the cause(s) of the changes – the aberrant genetic mechanism controlling brain development – may prove difficult to define.

Schizophrenia is still generally viewed as a 'functional' psychosis, a label implying a condition arising from disordered cognition with no basis in structural or pathological alteration of brain substance. Clinically, considerable emphasis is laid on the differential diagnosis of 'functional' psychoses from the dementias (chronic organic psychoses) in which cognitive and intellectual impairment are known to be related to structural changes in the brain.

Despite clinical evidence of brain dysfunction in schizophrenia, little progress was made in determining its organic parameters for most of this century and early neuropathological studies were conflicting and controversial (Corsellis, 1976; Roberts & Crow, 1987; Roberts, 1990). In a valedictory review, Corsellis (1976) sorrowfully summed up the lack of direction in the field and concluded that progress lay in the use of carefully matched samples and quantitative techniques to define the brain abnormalities underlying psychosis.

The current Lazarus-like state of research into the neuropathology of schizophrenia dates from the study of Johnstone *et al* (1976) which used the emerging technology of computerised tomography (CT) to re-examine the ventricular system in schizophrenics. They reported ventricular enlargement in schizophrenics which correlated with cognitive impairment but was unrelated to physical treatment. This study replicated earlier pneumoencephalographic studies (e.g. Lemke, 1935; Haug, 1962). The resultant flood of CT and, more recently, magnetic resonance imaging (MRI) studies have confirmed the finding (see Crow & Johnstone, 1987; Shelton & Weinberger, 1986) and lent credence to the idea of an underlying structural abnormality in the brains of many schizophrenic subjects, although the degree of ventricular enlargement observed is variable and not diagnostic.

This replicable evidence of structural abnormalities in the brains of schizophrenic subjects encouraged pathologists to adopt experimental approaches necessary for their detection (i.e. statistical comparison of quantitative data obtained from matched groups). Enough neuropathological data have now accumulated to attempt a synthesis with the epidemiological and genetic literature, which could provide a useful perspective for future research.

Brain structure in schizophrenia

Brain weight in schizophrenia has been studied since the beginning of the century, although the earlier studies are open to criticism on many grounds (Brown *et al*, 1986). Three recent studies have produced consistent results. Each found a significant decrease in brain weight in schizophrenic patients compared with controls after accounting for such factors as height, weight, sex, and year of birth. Brown *et al* (1986) reported an average reduction of some 100 g (6%), Pakkenberg (1987) a loss of 109 g (8%) and Bruton *et al* (1990), in a large prospective study, reported a 30 g loss from *each* hemisphere (approximately 5% of brain weight), and both male and female schizophrenics had reductions of a similar magnitude.

Other parameters of gross brain structure are also altered. Bruton *et al* (1990) found significant (4%) reductions (about 7 mm) in brain length (maximum anteroposterior length of formalin-fixed cerebral hemispheres) in both male and female schizophrenics. A separate analysis indicated that *both* left and right hemispheres were shorter. Pakkenberg (1987) found significant reductions in volume of the cerebral hemispheres (8% loss), cerebral cortex (12% loss), and central grey matter (6% loss). No loss in volume of white matter was found. No separate analysis of sex or laterality was made.

Post-mortem studies have also found evidence of ventricular enlargement. Brown *et al* (1986) reported a significant increase in the cross-sectional area of the anterior horn (by 19%) and temporal horn (by 97%) of the lateral ventricles, and Pakkenberg (1987) found an increase of 33% (10 ml) in the total volume of the ventricular system. Bruton *et al* (1990) in a blind semiquantitative study also noted a significantly increased incidence of ventricular enlargement in their schizophrenic sample.

While these data confirm structural abnormalities in the brains of schizophrenics, they provide little guidance as to which brain regions are particularly affected and how the structural abnormalities arise. The study of Pakkenberg (1987) indicates that cortical regions may be preferentially affected and data from Brown *et al* (1986) suggest that the ventricular enlargement may be regional in that the temporal horn is particularly affected. The enlargement of the temporal horn is lateralised, the left hemisphere being particularly affected (Crow *et al*, 1989).

Abnormalities in the gyral patterns of the temporal lobe have been described in schizophrenics (Jakob & Beckman, 1986) but the extent and precise nature of these sulcogyral abnormalities is uncertain and no such abnormalities were seen in the brains of schizophrenics in a larger, well controlled study (Bruton *et al*, 1990).

Brain regions affected

While CT and MRI studies are extremely useful for demonstrating structural changes in brains and allow 'fishing expeditions' for correlates of ventricular enlargement in schizophrenia, it should be remembered that larger ventricles are only an *indication* of pathology (an indirect one at that). The resolving power of such techniques is limited and detailed studies to pin-point specific regions and types of pathological change require painstaking and reproducible measurements on post-mortem tissue.

A planimetric study of archival material (the Vogt collection) revealed considerable volume reductions of temporal lobe structures (hippocampal formation, amygdala, parahippocampal gyrus) and a moderate reduction of the inner pallidal segment (by 20%) in schizophrenic subjects (Bogerts *et al*, 1985). The outer pallidal segment, caudate, putamen and nucleus accumbens did not show significant differences in volume.

In the largest planimetric study to date, Brown *et al* (1986) used subjects with affective disorder as a control group. All subjects (41 schizophrenics and 29 with affective disorder) were screened for senile or vascular change, and the findings were controlled for age, sex and year of birth. The brains of schizophrenics were 6% lighter (see above) and had enlarged ventricles. The schizophrenic group also had a significantly thinner parahippocampal cortex (11% reduction). No significant differences were noted in the areas of basal ganglia structures, third ventricle, cingulate cortex and insula opercula cortex. The thinning of the parahippocampal gyrus was significantly correlated with the increased area of the ventricular temporal horn (Colter, personal communication).

Thus both planimetric studies found evidence of structural change in the brains of schizophrenics, although to a lesser degree than that found in Alzheimer's disease or Huntington's chorea. Neither study provides support for the notion of homogeneous cortical atrophy or that the basal ganglia are particularly affected. Both conclude that the temporal lobe, specifically the medial temporal lobe structures (hippocampus and parahippocampal gyrus), are preferentially affected. The post-mortem studies are supported by a recent MRI study (Suddath *et al*, 1989) which used image analysis to measure the size of the temporal lobe and the amounts of grey and white matter in schizophrenics. Significant reductions were seen in the amount of temporal lobe grey matter (reduced by 18% in the right hemisphere and by 21% in the left hemisphere) in schizophrenics, particularly at the level of the amygdala and anterior hippocampus. These reductions correlated inversely with ventricular enlargement in the same sample. No differences were seen in the amount of temporal lobe white matter.

Medial temporal lobe structures (particularly the hippocampus) have been the subject of intensive study. Structural disorganisation of the hippocampal pyramidal cells has been reported (Kovelman & Scheibel, 1984). Disruption was maximal in the anterior and middle portions of the hippocampus (cf. Suddath *et al*, 1989, mentioned above) particularly at the interface between the entorhinal cortex and the hippocampus proper. These changes were considered suggestive of a defective pattern of neuronal migration. However, this finding of

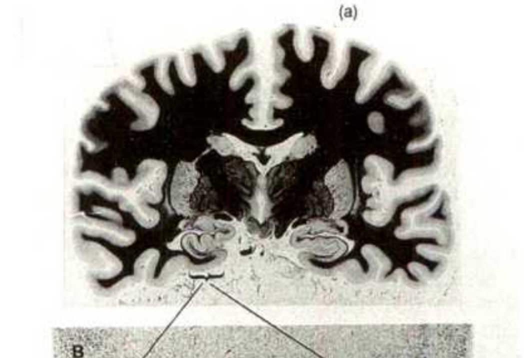

Fig. 1 Aberrant development in the parahippocampal gyrus in schizophrenia. The parahippocampal gyrus (PG) located close to the hippocampus (a) has a characteristic pattern of cell formations (b). In this region of cortex 'pre-αclouds' of neurons (cortical layer II, bracketed) lie close to the cortical surface. A high-power view (c) of one of the 'clouds' in a control (staggered line) shows it to be formed of two cell clusters separated by a cellular gap (dashed line). In schizophrenics (d) the pre-clouds are poorly developed, separate cell cluster not being discernible, and the whole formation lies deeper from the cortical surface (CS).

disorganisation has not been replicated in a more recent study (Christison *et al*, 1989).

Falkai & Bogerts (1986) reported that volumes of the hippocampal formation, pyramidal cell bands and hippocampal segments were decreased in the brains of schizophrenics. The absolute number of pyramidal cells was reduced and the loss of pyramidal cells was more noticeable in paranoid that in catatonic patients. The reduction of hippocampal nerve cells was not accompanied by gliosis (as indicated by counts of glial cells). In a further study on the same material (Falkai *et al*, 1988*a*) a reduction in both the area and neuron content of entorhinal cortex was found and, once again, the loss of neurons was not accompanied by increases in numbers of glial cells. These findings of decreased volume of the hippocampus and pyramidal band have recently been replicated (Jeste & Lohr, 1989). The decreased volume was most marked in the anterior hippocampus of the left hemisphere (cf. Crow *et al*, 1989).

A qualitative description of morphological changes in the temporal lobe of schizophrenics has been presented by Jakob & Beckman (1986). The changes include alterations to and cell loss from cortical lamina of the ventral insula (claustrocortex) and parahippocampal gyrus. The medial and central portions of the parahippocampal gyrus were most disturbed, with the lateral (transitional) region less affected (Fig. 1). These changes did not extend anteriorly to the pre-piriform cortex nor posteriorly to the occipital level of the temporal cortex (cf. Kovelman & Scheibel, 1984; Suddath *et al*, 1989). The cytoarchitecture of the remaining temporal neocortex was normal. The cytoarchitectural abnormalities were present in *both* hemispheres but more conspicuous in the left hemisphere. Of interest was the tendency for patients with marked pathological abnormalities to have an earlier age of onset of psychotic symptoms. These findings were considered to indicate abnormal ontogenetic development of a small part of the entorhinal cortex. This study is open to some criticism; it was not blind, not quantitative and only ten control cases (as opposed to 64 schizophrenic cases) were assessed. However, a preliminary quantitative study of the cytoarchitecture of the pre-alpha cells of the parahippocampal gyrus revealed evidence of putative migration anomalies (Falkai *et al*, 1988*a*; Fig. 1c,d).

Other brain regions can be affected to a lesser degree (see Roberts & Crow, 1987; Roberts, 1990; Roberts & Bruton, 1990).

Neuropathological studies of structural changes and clinical symptoms

Cognitive impairment (and associated negative symptoms) and measurements of poor pre-morbid and social functioning appear to be best related to loss of brain weight, decrease in brain length, increased ventricular size and abnormalities in cytoarchitecture (Jakob & Beckman, 1986; Pakkenberg, 1987, Bruton *et al*, 1990). In general, the greater the structural changes, the greater the cognitive impairment and the poorer the pre-morbid function. Cognitive impairment and poor pre-morbid function are associated with early onset of the illness and a poor prognosis (Shelton & Weinberger, 1986; Crow & Johnstone, 1987).

While the correlations between measures related to cognitive impairment (negative, or type II symptoms (Crow, 1980)) and structural changes is discernible, it should be remembered that many of these patients also have delusions, hallucinations and incongruity of affect (positive, or type I symptoms). Thus, studies in this field are at an early stage and the conclusions concerning the clinical correlates of altered morphology should be regarded as preliminary.

The clinical state of schizophrenia has been viewed as a common endpoint for a number of discrete aetiological factors. Various studies have sought to relate measures of structural abnormality (usually ventricular enlargement) to subgroups of schizophrenics defined on the basis of symptoms or presumed aetiological factors (Crow, 1980; Murray *et al*, 1985; Lewis & Murray, 1987; Lewis, 1989*a,b*). None of these attempts has been entirely convincing. Despite the proposal that ventricular enlargement might be confined to patients with negative symptoms or a history of birth difficulties, it has in fact been found in patients with or without positive symptoms, negative symptoms, and with varying degrees of assumed genetic loading (Farmer *et al*, 1987; Nimgaonkar *et al*, 1988; Owen *et al*, 1989).

Murray & Forester (1987) hoped that psychiatrists of the future would be able to define subgroups or types of schizophrenia "according to aetiological principles rather than on the quicksands of symptomatology and course". At present there is no consistent evidence that a rationale subgrouping schizophrenics on the basis of the presence or absence of structural change or other aetiological factors can be made. Thus, it seems simpler to assume that *all* schizophrenics have a degree of structural abnormality and that, neuropathologically, schizophrenia is best viewed as a homogeneous condition: the symptoms of schizophrenia arise from a type of lesion which differs in degree but not kind.

The origin of the structural changes

Kraepelin (1919) originally conceived of schizophrenia as a degenerative process. This was modified in later years by others to a series of variations of stress-diathesis models and psychobiological theories. The early CT findings were regarded as evidence for an underlying degenerative process and focused attention back to Kraepelin's early ideas. Schizophrenia was again viewed as a degenerative process (possibly caused by an acquired pathogen) (e.g. Crow, 1983). This view fitted with the correlation between ventricular enlargement and cognitive impairment, a finding to be expected in a neurodegenerative condition. The report of increased periventricular gliosis (Stevens, 1982 – but see below) was also in accord with a "low-level of viral infection". However, the correlation between length of illness and increasing ventricular size seen in other neurodegenerative diseases failed to emerge (see Weinberger, 1984, 1987; Crow & Johnstone, 1987). Furthermore, changes of a similar degree were found in patients only weeks or months after the onset of their first psychotic episode. In view of this background it was hypothesised that schizophrenia was of multifactorial aetiology, with family history (genetic factor) and obstetric or perinatal complications (environmental factor) being regarded as the most prominent (Murray *et al*, 1985, 1988).

Several recent studies have attempted to obtain evidence of the 'smoking gun' which might implicate one of these factors directly. However, identifying aetiological constants in schizophrenics in pathological studies is made more difficult by the findings from neuropathological surveys, which show that, as a group, schizophrenics have a high incidence of pathological changes (e.g. infarcts, vascular disease, Alzheimer-type changes, Parkinson's disease, etc.): Stevens (1982) had found pathology in 50% of cases, Jellinger (1985) in 51%, and Bruton *et al* (1990) in 43%. In the latter study, the incidence was significantly greater ($P > 0.01$) than the 21% incidence found in age- and sex-matched controls, although the *types* of pathology found are qualitatively indistinguishable. The explanation for these observations is unclear, although several possibilities have been canvassed (Bruton *et al*, 1990), such as lifestyle, drug treatment, selection bias and, more interestingly, a secondary vulnerability to damage due to underlying structural changes.

Many neuropathological studies have focused on gathering evidence for the presence of a neurodegenerative process and ascertaining when the structural changes occurred. All types of disease process which cause neuronal damage result in glial reaction, which is marked by proliferation of glial cells and rises in associated glial proteins and enzymes (such as glial fibrillary acidic protein and monoamine oxidase-B) (Roberts *et al*, 1987). Finding a constant pattern of such reactivity in schizophrenic brains would signal a neurodegenerative process and indicate the location of the damage.

Stevens (1982) reported periventricular gliosis in schizophrenics. However, as noted above, some 50% of her cases had evidence of identifiable pathology. Bruton *et al* (1990) also noted an increased incidence of gliosis in the schizophrenics, but this was significantly associated with the various types of pathology present in the sample. Further analysis showed that the application of a neuropathological 'sieve' to the total sample (removing cases with identifiable pathology excluding gliosis) gave a 'purified' group which still showed reduced brain weight, brain length, and increased ventricular size but no significant gliosis. Recent quantitative studies using similar selection criteria and a variety of methods report similar results (see Roberts & Bruton, 1990). The available pathological evidence indicates that the structural changes in the brains of schizophrenics are not the result of a neurodegenerative process or a destructive lesion. Rather the data fit the profile of a disturbance in brain development (Roberts *et al*, 1987). Although ideas of maldevelopment in schizophrenia have a long history (see Lewis, 1989) it is only recently that such a concept has been firmly rooted in data obtained from direct observation of the neuropathology of the condition.

The idea of schizophrenia as a neurodevelopmental anomaly of the medial temporal lobe gives rise to several predictions (Roberts *et al*, 1987). The lesion would be present from birth, preceding the onset of clinical symptoms and may be constant and not progressive. Only a few studies have addressed these questions. Ventricular enlargement has been reported in a ten-year-old diagnosed as schizophrenic (Woody *et al*, 1987) and in two teenagers scanned *before* the onset of schizophrenic symptoms (O'Callaghan *et al*, 1988; Weinberger, 1988). There is little or no evidence of progressive structural changes. In the first such study to examine the problem only 20% of patients were reported as showing progressive ventricular enlargement over four years (Lemke, 1935) and three recent studies show no evidence of progression in groups of patients scanned over eight years (Illowsky *et al*, 1988), and five years (Vita *et al*, 1988; Reveley *et al*, 1988).

Neuropathological studies and suggested aetiological factors

Are the neuropathological data compatible with the intrusion of external environmental events (such as those caused by obstetric complications or infection) on brain development around the time of birth?

Foetal brain tissue does not show an adult glial response until after six months (Larroche, 1984) and an early destructive lesion might not leave a gliotic trace. However, the type of changes to the cytoarchitecture reported in the schizophrenic brain (Jakob & Beckman, 1986) indicate that they originate in the last trimester before birth, a period when an adult-like glial response to injury does occur (Larroche, 1984; Volpe, 1989). Birth difficulties can give rise to intraventricular haemorrhages and infarcts which lead to enlarged ventricles and small brains (Gilles & Green, 1985; Volpe, 1989). Such pathologies *are* accompanied by gliosis and periventricular leucomalacia. However, neuropathological studies report no excess gliosis and an absence of inflammation (Roberts *et al*, 1986, 1987; Bruton *et al*, 1990) and there is little evidence of trauma (Bruton *et al*, 1990). These data do not suggest that brain damage due to birth injury or perinatal infection is a major factor in the genesis of the structural abnormalities in schizophrenia. Thus, direct evidence for the involvement of external environmental events in the genesis of schizophrenia is lacking.

The argument that external environmental events cause the changes in brain structure in a majority or significant minority of schizophrenic cases rests on a number of pillars: (a) the lack of 100% concordance for schizophrenia in monozygotic twins, (b) the excess of winter births in schizophrenics, (c) the increased incidence of birth complications in schizophrenics, and (d) the occurrence of schizophrenic symptoms in various neurological conditions. This evidence has been extensively reviewed (Davison & Bagley, 1969; Murray *et al*, 1985; Crow & Johnstone, 1987; McGuffin *et al*, 1987*b*; Lewis, 1989; Roberts *et al*, 1990). However, most of these pillars have recently met their experimental Samson and the details of these encounters are worth briefly noting.

The determination of heritability based on monozygotic or dizygotic concordance rates may no longer be valid in general (Burn & Corney, 1988) and may only represent the lower limit of heritability in schizophrenia in particular (Gottesman & Bertelsen, 1989). Monozygotic twins discordant for the expression of Duchenne muscular dystrophy have been reported (Burn *et al*, 1986). To explain this phenomenon it has been proposed that a single egg can form clones at the blastocyst stage and that these clones (the twin pair), although genetically identical, will differ in their expression of the aberrant gene (Burn *et al*, 1986). Such a phenomenon could explain some of the discordance for schizophrenia in monozygotic twins (see Gottesman & Bertelsen, 1989). The excess of winter births accounts for about 5% of cases and this may be an overestimate since it has been argued that reanalysis of the original data on winter births reveals the possibility of an ascertainment artefact which might diminish the significance of the effect (Lewis, 1989,*a*,*b*). On the basis of current data, birth complications might represent a significant aetiological factor in 1–2% of

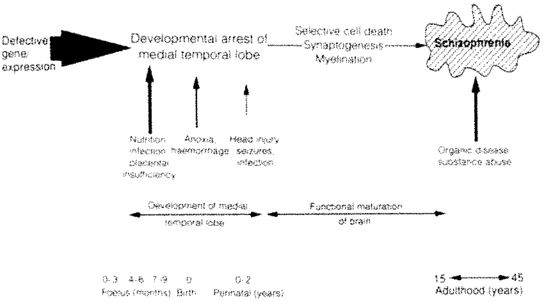
Defective gene/ expression
Developmental arrest of medial temporal lobe
Selective cell death
Synaptogenesis
Myelination
Schizophrenia
Nutrition infection placental insufficiency
Anoxia, haemorrhage
Head injury seizures, infection
Organic disease substance abuse
Development of medial temporal lobe
Functional maturation of brain
0-3 4-6 7-9 0 0-2
Foetus (months) Birth Perinatal (years)
15 45
Adulthood (years)

Fig. 2 The relative importance of different factors in the aetiology of schizophrenia (size of arrow reflects relative importance of factor).

cases of schizophrenia. However, the robustness of this finding and the direction of causality (i.e. do birth complications cause schizophrenia or does the expression of the schizophrenia genotype cause birth complications?) has been questioned on epidemiological grounds (Goodman, 1988). Psychotic symptoms indistinguishable from schizophrenia occur during various kinds of neurodegenerative disease (Johnstone *et al*, 1988), notably Alzheimer's disease (Drevets & Rubin, 1989) which particularly affects pre-alpha cells in the medial temporal lobe. However, the incidence of unsuspected organic disease in schizophrenics is only about 5% (Johnstone *et al*, 1987).

Even if taken at face value, the identified environmental factors could account for some 15% of schizophrenia cases. However, bearing in mind the criticisms noted above, 15% may well be an overestimate of cases resulting from environmental factors. Overall the evidence from neuropathological and epidemiological studies supports the view that a developmental anomaly causing structural changes in the brains of schizophrenics is likely to be *genetic* in origin in the majority of cases. Environmental factors such as birth complications or other organic disease which could give rise to phenocopies of schizophrenia are probably responsible for a small, possibly insignificant, number of cases (Fig. 2).

Normal brain development and structural changes in schizophrenia

The human brain develops asymmetrically (Chi *et al*, 1977). The gyral pattern of the temporal lobe develops from the 31st week to term, with the left hemisphere developing one to two weeks *later* than the right. The hippocampus appears during the latter half of pregnancy and reaches approximately 50% of the adult volume by birth. The adult volume is reached at about two years of age. The hippocampi in each hemisphere are symmetrical both in size and pattern of growth (Kretschman *et al*, 1986). The hippocampus forms the medial wall of the temporal horn of the ventricular system. During brain development ventricular size (as a percentage of brain area) is *reduced* (Koop *et al*, 1986). This is particularly noticeable in the temporal horn, whose cross-sectional area is halved between birth and nine years of age (Kurihara *et al*, 1986). So the volumetric growth of the medial temporal lobe causes a compression of the ventricular system, particularly the temporal horn.

The development of the parahippocampal gyrus is asymmetrical and earlier than the development of the hippocampus. The two structures are functionally connected by the perforant path, an excitatory projection from the parahippocampal gyrus/entorhinal cortex into the hippocampus. Loss of or alteration to projection systems during *development* can result in aplasia or dystrophy in the target structure (in this case the hippocampus) (see Roberts, 1990).

The pattern of normal brain development provides an explanation for the structural changes seen in the brains of schizophrenics. The mechanism causing the aberration in brain development is effective during the third trimester, it affects both hemispheres, and it alters the development of the temporal cortex, particularly the parahippocampal gyrus. Since development of the left temporal cortex lags behind the right, any factor which simultaneously arrests both will inevitably affect the left more than the right.

Thus the asymmetric changes in the parahippocampal gyrus will give rise to asymmetrical effects on the (usually symmetrical) development of the hippocampus. This in turn could produce asymmetry of the temporal horn. Suddath *et al* (1989) presented evidence for reduced volume of the temporal lobe, which was inversely correlated to the area of the temporal horn (cf. thinning of parahippocampal gyrus and increased temporal horn area from study of Brown *et al* (1986), discussed above). They argue that changes in the volume of the temporal lobe are translated into relatively larger changes in ventricular volume. Thus ventricular volume acts as a sensitive indicator of reduced brain volume.

Anatomical significance of the parahippocampal gyrus

Information from the association cortex (temporal, cingulate and frontal cortices) converges on the entorhinal cortex and reaches the hippocampus via

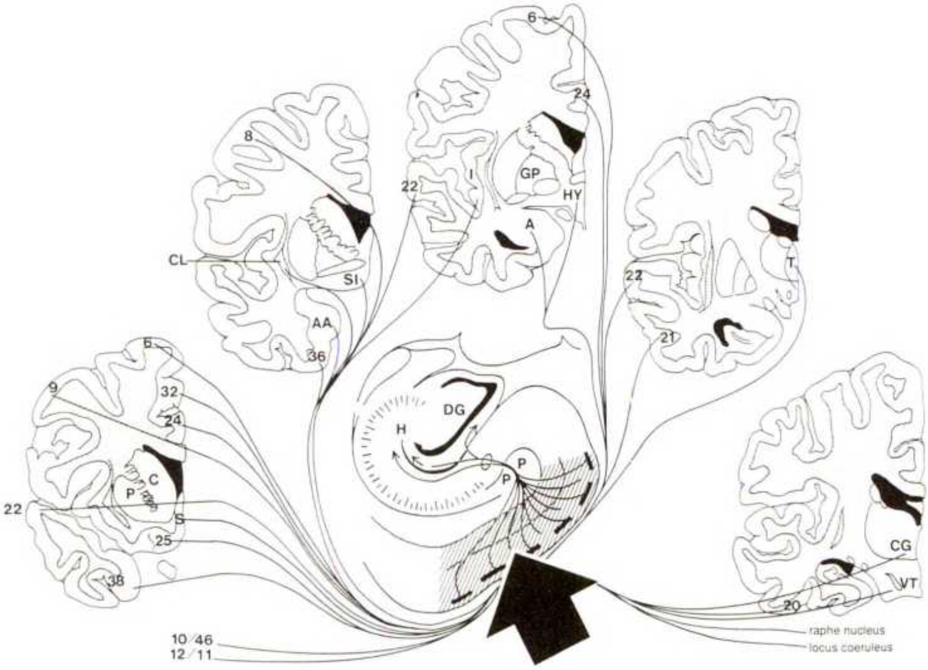

Fig. 3 Projections to the parahippocampal gyrus. The links between various cortical and subcortical areas and the parahippocampal gyrus are shown diagrammatically. The projections shown are those from the same hemisphere but it should be noted that similar (but smaller) projections from the *opposite* hemisphere are also present. The terminal regions of the projections are topographically organised and often separate projections arise from subdivisions of one region (e.g. amygdala). The diagram is a synthesis of data obtained from Van Hoesen (1982), Amaral *et al* (1987) and Insausti *et al* (1987*a*,*b*). (▨ parahippocampal gyrus, ▩ white matter, ■ ventricle, A, amygdala, AA, anterior amygdala, C, caudate, CG, central grey, CL, claustrum, DG, dentate gyrus, GP, globus pallidus, H, hippocampus, HY, hypothalamus, I, insula, P, putamen, PP, perforant path, S, septum, SI, substantia inominata, T, thalamus, VT, ventral tegmentum, 6–46, Brodman areas.)

polysynaptic relays in the parahippocampal gyrus and subiculum (Van Hoesen, 1982; Insausti *et al*, 1987*a*,*b*)) (Fig 3). Hippocampal output is organised in a similar fashion. By virtue of this arrangement the parahippocampal gyrus and subiculum serve to 'gate' all information entering and leaving the hippocampus. Direct connections link the medical temporal cortex and the frontal cortex, striatum and amygdala. Temporal cortex, hippocampus and amygdala are thought to be central in the integration of affect and intellect (Frith & Done, 1988). Changes in the functional ('gating') capabilities of the parahippocampal gyrus could have a significant effect on such integrative processes and have large effects on the functioning of many other systems (e.g. dorsolateral pre-frontal cortex) (Weinberger, 1987; Frith & Done, 1988). Changes in the structure and function caused by the developmental abnormalities will inevitably alter the neurochemistry of other brain regions. Neurochemical changes in the brains of schizophrenics are lateralised to a certain extent and have been reviewed (Reynolds, 1989).

The brain in schizophrenia – a summary

Neuropathological studies indicate that the brains of schizophrenics suffer from a developmental abnormality which preferentially, but not exclusively, affects the medial temporal lobe (parahippocampal gyrus, hippocampus and amygdala). Medial temporal lobe structures are believed to have a crucial role in the integration and processing of the input from association cortex. Dysfunction of this system could result in the clinical symptoms that form the core of the schizophrenia syndrome. The cause of the anomaly is probably genetic in the overwhelming majority of cases and affects brain development in the third trimester. The asymmetrical pattern of development in the normal human brain explains why such a defect affecting both hemispheres simultaneously will disproportionately affect the left (dominant) hemisphere.

Despite the great progress made in determining the nature of the structural changes in the brains of schizophrenics, the cause of the changes – its mechanism – is still the subject of speculation.

Factors relating to the mechanism of structural change

The evidence for a genetic component of schizophrenia has been exhaustively reviewed (e.g. McGuffin *et al*, 1987*b*) and it is accepted that while there is a genetic component of schizophrenia, its nature (single gene with variable penetrance, polygenic, etc.) is obscure.

Discrete possibilities about genetic mechanisms are difficult to discern. Two classes of genes have been proposed to be involved in cortical development (Rakic, 1988). One class is thought to regulate the number of radial units (columns) in the cortex. A failure here would reduce the number of columns but the neuronal content *within* each column would remain normal. These defects would be early events in corticogenesis (in humans within six weeks of gestation); reduced numbers of proliferative radial units are established in the ventricular zone but each unit can produce the normal (genetic) number of neurons. Cortex suffering from such defects would have a smaller surface area in spite of normal or enlarged thickness and significant neuronal ectopias (as seen in lissencephaly and pachygyria). This is *not* seen in schizophrenics.

A second class of genes is responsible for the control of neuronal proliferation within radial units. Defects in these genes might cause diminished neuronal production within radial units, giving fewer neurons in ontogenetic columns and therefore a thinner cortex. Neuronal number in ontogenetic columns could also be affected by programmed cell death or migrational failure. Thinning of the parahippocampal gyrus is seen in schizophrenia and this second class of genes may repay close study.

Despite this pointer, the complexity of the genetic regulation of brain development is only just becoming apparent (Nowakowski, 1987; McConnell, 1988; Rakic, 1988). Genes on different chromosomes affect the same cell types, other single genes affect widely different cell types and feedback mechanisms exist between afferent and target cells which determine cell survival and regulate synaptogenesis. Overlaying these mechanisms is the crucial factor in development – that of timing. Widely disparate mechanisms (different genes or perturbations in gene expression) operating at the same stage of development produce similar results. Epidemiologically, few cerebral malformations appear to be caused by simple Mendelian inheritance (Larroche, 1984). The fine processes that shape brain development could be viewed as an example of a chaotic phenomenon, where minor changes in any one of dozens of genes will have unpredictable effects. In such circumstances arrest or curtailment of cellular migration is more frequent than complete failure and such phenomena may be more common than previously realised in normal human brains (e.g. see Kaufman & Galaburda, 1989).

In schizophrenia the focus of the abnormality is the parahippocampal cortex and this stone in the developmental pond causes ripples which can spread to affect other brain areas. The magnitude of the genetic defect and the timing of its expression are the vector that determines the size and spread of the ripples. The ripples affect the dynamics of development and cause long-term defects in intellectual function as the brain completes its processes of maturation via mechanisms of programmed cell death, synaptogenesis and myelination (Schneider, 1979; Weinberger, 1987; Goodman, 1989) (Fig. 2). Thus a chaotic parahippocampal tail grows up to wag the temporal dog.

The magnitude of the task of untangling the mechanisms that contribute to the schizophrenogenic brain is only now becoming apparent. Many genes could contribute to the structural abnormalities outlined above and none may be more important than the others. Future studies to locate the gene for schizophrenia might find it useful to bear in mind this background and recall the hard-won lessons on the multifactorial genetics of predisposition to coronary heart disease and thus avoid schizophrenia becoming the mausoleum of molecular biology (see Lander, 1988; Mullan & Murray, 1989; Robertson, 1989).

However, the speed of progress in neuropathological investigations allied to the expansion in our understanding of the genetics of developmental neurobiology augurs well for the future and offers a way ahead. Neuropathological studies should aim to define the essentials and limits of structural changes (e.g. cell types affected and altered patterns of neuronal connections) and relate these to the data on factors and mechanisms responsible for the regulation of such phenomena derived from basic studies of brain development. In this way the complexities of genetic regulation can be reduced to postulate mechanisms (target genes) and suggest practical ways of pin-pointing which aberrant genetic structures or regulatory mechanisms generate the brain abnormalities that cause schizophrenia.

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Self-esteem as a Factor in Social and Domestic Violence

ISMOND ROSEN

Psychoanalytical ideas are used to outline how poor narcissistic development can lead to poor regulation of self-esteem. This in turn often underlies domestic violence, the short-term treatment of which can be aided by psychodynamic understanding.

Growing public intolerance of adult domestic violence has resulted in significant changes in official attitudes. A recent Home Office circular provided guidelines to the police to regard assaults in the home as seriously as other forms of violence, to keep detailed records of such incidents, and to avoid attempts at reconciling partners where a quarrel had come to blows (reported in *The Times*, 1 August 1990). Irate British husbands may no longer punch their wives on the nose with impunity, nor subject their wives to forcible sex. "In Streatham, London, the police have been operating an arrest-and-caution policy for two years. More than three-quarters of those cautioned have not re-offended. To be seen by neighbours taken out of the house by the police is a humiliating experience" (apart from an uncomfortable night in the cells and even the risk of prosecution) (*The Times*, 1 August 1990).

Sir Peter Imbert, the Commissioner of the Metropolitan Police, in a lecture to forensic psychiatrists, described two main courses of action by the police – "an interventionist approach, by way of arresting the offender, and a much enhanced support and consideration for the victim" (Imbert, 1990). The police now assist victims by referring them to aid societies for support, for counselling, or to places of refuge. Evidence for the success of this approach comes from Canadian research, where police intervention significantly reduced re-offending, in that the assailant was given a clear message that his behaviour was unacceptable and would not be tolerated.

Acts of social violence are usually the manifestation of complex intrapsychic and interpersonal behaviour in which aggression, drive control, and a sensitive self-esteem interact. The painful devaluations of the self which trigger and result from violence are often found to be repetitions of earlier childhood experiences. It is interesting to note that the police interventionist approach may provide a deterrence in those already lowered in their self-esteem. Where such 'short sharp shocks' fail, one would infer that their self-esteem systems are seriously disturbed, with faulty controls and unconscious compulsions to act violently.

This paper outlines psychoanalytical concepts of self-esteem and its regulation, drawn from many schools, to illustrate how such dynamic understanding may be brought to bear in initial encounters or the short-term treatment of social violence, especially marital disharmony.

One of the earliest recorded examples of the relationship between self-esteem and social violence is that of Moses smiting an Egyptian who was being cruel to a Hebrew. The Bible significantly relates how the next day Moses interceded between two Hebrews who were smiting each other, whereupon one of the men asked Moses "Who set you up as a judge over us?" (or, as we would say nowadays, "Who do you think you are?"). The man then asked, "Will you do to us what you did to the Egyptian?" Moses took fright because his violence, which he thought went unnoticed, was now known, and he escaped into hiding to avoid the wrath of Pharoah.

At the time of smiting the Egyptian, Moses must have had some unconscious conflict of identity. Was he Hebrew or Egyptian? Moses' violence was in the service of an ideal – that of rescuing and avenging a fellow Hebrew – but he had been raised in the royal Egyptian household, and he had acted with the omnipotence of an archaic prince. Acts of violence often express a coded statement of identity – "How dare you devalue me. I'll show you who I really am." If one adopts a particular orthodox view of Moses as perfect, one must regard his violent action as one taken purely on moral grounds, resulting from his mature Hebrew judgement about a cruel man. But the Biblical details, like the brush strokes in a painting, reveal a more complicated underlying psychic identity, in which Moses undergoes a process of maturity in the development of his narcissism and regulation of self-esteem.

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In modern psychoanalytical parlance, as described by Kohut (1973), Moses' action was evidence of his grandiose self, or immature ideal self, to which he had recourse as the result of a narcissistic rage, in which immediate revenge was achieved by turning an experience of cruelty to a Hebrew slave, with whom he passively identified, into an active attack on the perpetrator. Moses simultaneously identified unconsciously with the cruel aggressor and became oblivious of the consequences of his actions. We could therefore ascribe Moses' action as the result of an admixture of his lowered self-esteem resulting from his identification with the Hebrew slave, and his regression to violence based on his omnipotent self. His self-esteem was further lowered next day by the questioning by the Hebrew "Will you do the same to us?", which inferred that he was a man of violent character, rather than an idealist. Shame and guilt ensued and Moses escaped into the land of Goshen.

Shortly after this, Moses appears as a more diffident character, who stammers and requires his brother Aaron to represent him. His rage surges forth again when he breaks the tablets of the law on Sinai. We revere Moses the Lawgiver for having been given the Ten Commandments, including "Thou shalt not kill", but Moses remained a man, humbled and punished by the Lord for the Israelites' lack of faith at the Waters of Meribah, for which he was not allowed to enter the Promised Land. From the point of view of self-esteem, the saga of Moses can be described as a process of narcissistic maturation, where his princely, Egyptian, omnipotent archaic self matured into an ideal self which was harnessed in the service of a Hebrew divinity and people. The authority of Moses' grandiose self had been given up in favour of the ideal object and his subservience to the law – the very image of the perfect man.

Psychoanalytical theory of the regulation of self-esteem

Self-esteem is defined by the *Oxford English Dictionary* as "an appreciation of one's self". Psychoanalysis currently defines the 'self' as 'self-experience', 'sense of self', or 'self-image'. Used in these ways, the self has to be differentiated from self-representations, which result from the psychic process of internalisation of the residues of self-experience. These self-representations exist as internal objects or structures within the mind. Such self-objects are endowed with energy or emotional interest drawn from libidinal and aggressive drives. The process by which this investment takes place in earliest life is known as infantile narcissism. Briefly speaking, the better the childhood rearing experiences, the richer the inner self-objects and the more self-reliant and confident the personality. One of the factors contributing to the development and regulation of self-esteem was the individual's perception of his/her own worth in the eyes of parents or parental figures, in a process known as 'mirroring'.

All of us attempt to maintain our level of self-esteem within a given range. To counteract feelings of devaluation, some individuals will resort to any means to redress the balance, such as the recourse to physical action or aggression, by sexual stimulation, alcohol, drugs or risk taking. The regulation of self-esteem has been described in depression by Jacobson (1975), and in perversion by Rosen (1979). One may assume the presence of a monitoring mental agency which regulates the level of self-esteem, defending the self against devaluation from whatever source, conscious or unconscious, external or intrapsychic. Should self-esteem suddenly fall or be threatened, trigger mechanisms operate, in those susceptible, like computer programs on a stock exchange, which execute orders spontaneously, sometimes leading to chaos. Violent behaviour may therefore be triggered by a narcissistically wounding experience, or the sudden lowering of self-esteem below a critical level in a predisposed person, hitherto blameless in this regard.

Violent behaviour, like sexual behaviour, is extremely protean in manifestation and has its precursors in infantile experience. The frustrations inherent in infantile life lead to the development of an immature ideal self which is endowed with feelings of omnipotence, hence Kohut's use of the term indicating a separate agency in the mind – the ideal or grandiose or omnipotent self. This early narcissism and self-love develops into a more mature agency, the ego ideal.

Much domestic violence results from slights which threaten the assailant's ego ideal and lowers his/her self-esteem. The prevailing attitude becomes one of 'How can you do this to me?' Violence may then occur as a result of the individual's regression to the infantile omnipotent grandiose self in those predisposed, with the need for physical aggression to raise self-esteem. The violent act may take place against the conscious will of the person, even where other real satisfactions are open to them. Relationships with spontaneously violent persons are constantly jeopardised by any attitude or events which they perceive as a threat to their ego ideal and self-esteem. On the other hand, those who compulsively seek revenge may act with deliberate, cold, calculated cunning.

Among the many factors in child rearing leading to a healthy self-esteem, the most significant is where the child's self-accomplishments are promised and accurately extolled and mirrored by the parents. Other factors include the child's freedom and opportunity for imitation and identification, both in the early mother–child relationship and the later relationship with the father, which ensures that same-sex identifications are facilitated. Healthy ego-ideal development proceeds where the family sets goals for achievement by the child that are attainable.

Ego-defence mechanisms in the child are partly based on identification with parental defence mechanisms. Attitudes and overt expression of violence may be transmitted this way.

Narcissism and the narcissistic personality

A certain amount of narcissism or self-interest is normal and desirable. Narcissism may range from a feeling of self-worth and self-confidence to feelings of inadequacy and worthlessness. Psychologists have tried to determine scales where normal narcissism ranges into the excessive and seriously disordered: Raskin & Hall (see Emmons, 1987), constructed a Narcissistic Personality Inventory (NPI) based on DSM–III criteria for the narcissistic personality disorder. The NPI measured individual differences in narcissism as a personality trait. Five factors appeared significant, which when manifested to an extreme degree, constituted pathological narcissism. These were as follows:

- (a) a grandiose sense of self-importance and uniqueness
- (b) the preoccupation with fantasies of unlimited success or beauty
- (c) exhibitionism, requiring constant admiration and attention
- (d) an attitude of entitlement, with the expectation of special favours from others without the need for any reciprocation
- (e) interpersonal exploitativeness.

The distinction between a normal personality trait and pathological narcissism would be one of degree. There is ample evidence clinically and in the literature that violent people suffer from disorders of narcissism. In the narcissistically disordered, acts of violence may be triggered by sudden diminutions of self-esteem, because in such individuals there is psychic access and regression to an immature, archaic, grandiose self.

Three major unconscious defence mechanisms are especially operative in violent individuals. The first is *projective identification*, where unwanted aspects of the self are projected externally on to others. One man who suffered from a state of chronic narcissistic rage tried to be pleasant to his wife and family, but had occasional outbursts. Their child, however, would abuse the mother at the least provocation, and became the 'monster' of the family. This child's rage against his mother was a projection of his father's repressed rage against his own mother. When the father improved in treatment, the boy became more manageable. Two other defence mechanisms are invariably present.

Turning active into passive is a mechanism which underlies the generational continuity in families. Patterns of violent behaviour or physical humiliations experienced passively in childhood are later enacted by the adult against his/her children or spouse, who is unconsciously identified with the original offending parent, whatever the sexual identity. Thus to an assailant a wife victim could stand for a punitive mother or father. For the victim, the assailant may be unconsciously identified with the parent of either sex, and to whom they remain compulsively attached.

The mechanism of *identification with the aggressor* enables patterns of violent behaviour which are not experienced personally to be copied or stored preconsciously. These pre-formed imitative patterns of violence may be spontaneously triggered by esteem-lowering experiences, or the need for revenge. A unique act of violence in one country may be copied as a result of international media publicity. Football violence is no doubt influenced in this way.

Narcissism and early bodily development

Formative experiences of the self in relation to others determines our later vulnerability to stress and the variability of our self-esteem. Kohut has stressed that narcissism, or self-cathexis, is not a dynamic counterpart to object-cathexis on the model of the fluid in a U-tube, but is a distinct part of the psyche with its own developmental sequence and maturational pathway.

Individuals who have lacked confirmation responses (mirroring) from their parents may come to depend on their own bodily self-stimulation to maintain self-esteem. This may appear in childhood in severe cases as the self-infliction of pain such as head-banging, or sexually as compulsive masturbation. Bodily stimulation enables them to retain a sense of aliveness and personality cohesion. One patient who had been regularly battered as a child and experienced very little mirroring from his mother resorted to bouts of violence on appropriate provocation. As an indirect result of successful analysis he was deeply grateful when his lifelong habit of compulsive masturbation disappeared. In the transference he relived very early bodily wishes to be touched, at which times he felt in danger of a psychic breakdown. He had been a compulsive masturbator since childhood, and it was only when this element was removed that he felt forced to conclude that he was being helped in therapy and that his real self had at last been reached.

The regression to early body-ego needs is one of the common meeting grounds between violence and the sexual perversions, where both are an expression of defence against lowered self-esteem and depression. Perversion usually defends against violence. Where violence occurs in a sexually perverse setting, it is because the libidinal sexual elements have failed to contain a build-up of tension that has its final expression in violence. Overt violence in both assailant and victim may be the means of avoiding a more dangerous build-up of tension that threatens a break-up of the assailant's or the victim's self and may promote his/her self-cohesion. The provocativeness of the victim in the battered-wife syndrome may have a defensive function against object loss. Some battered wives leave their husbands only when they are made to feel ashamed by their children.

Body-ego deficiencies, narcissism and rage reactions

The sensitivity to narcissistic slights may originate in a sense of body-ego or organ inferiority. The awareness of a damaged body part or deficient function may be split off or denied by omnipotent ideas. A breaching of the denial and a reinforcing of the awareness of the body defect may produce shame and rage, usually of a verbal kind. The repression of such rage in those born physically handicapped may be associated with psychosomatic symptoms or skin disorders.

The experiences of infantile genital inadequacy during the oedipal and phallic phases in both sexes may of course contribute to and complicate the clinical picture of narcissistic sensitivity.

Narcissistic rage

Narcissistic rage as described by Kohut is part of a continuum, from fleeting annoyance to the unbridled furore of some murderers. It is the fight aspect of the biological substratum of the fight/flight response.

The expression of narcissistic rage is usually found in the setting of revenge. In terms of their ideals, such persons are righting a wrong or undoing a hurt, which they will pursue relentlessly by any means. There is an utter disregard for any limitations or consequences of their actions and there is a total lack of empathy for the victim or person who triggers the particular rage response. These qualities are based on the inner notion of the perfection of the idealised self-object. The narcissistically injured person cannot rest until he has wiped out the opposition. More importantly, narcissistic rage may arise when the self or the object fails to live up to the asbolute or ideal expectations which are made of them. In my experience, some individuals may compensate for the poverty of their real inner selves and low self-esteem by their social and intellectual accomplishments. But they remain sensitive and vulnerable to outbursts of anger and may rage in great distress in private.

Although everyone tends to react to narcissistic injuries with embarrassment and resentment, the most intense experiences of shame and the most violent form of narcissistic rage arises in those narcissistic individuals whose absolute control over their environment is threatened. The maintenance of their self-esteem depends on two main sources: the unconditional availability of other persons as admiring objects, or the ever-present opportunity for a merger or identification with an idealised object. Where previously the deity, the nation and royalty reigned supreme, these have been replaced by riches, pop stars, football teams or political dictators.

People with narcissistic sensitivity are vulnerable in several ways. They are more at risk of intimate violence than others, particularly at times of stress. Emmons (1987) has confirmed earlier findings that narcissistic individuals also suffer mood swings and extremes of affects. Some optimism is possible however, in that personality maturation and the complex growth of the self may occur as a result of marriage or by exercising leadership, so that the effective responses become more stable.

Domestic violence and wife battering

Domestic violence can be considered as an aberration of the process of intimacy. The capacity for intimate living is maturational and is developed into adulthood, beginning with the infantile mother–child and later family relationships. The personality distortions arising from such disturbances of those early experiences vital for the development of the capacity for intimacy are the same as those factors pertinent for the manifestation of adult violence. Traumas associated with the failure of intimacy experiences in adolescence or adult life may reinforce the vulnerability to violence. The regression to earlier disturbances of intimacy at specific phases in childhood is seen in wife battering, for example, which begins

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at one of three critical stages of intimacy in the marriage.

The first occurs shortly after marriage, where the domestic violence literature refers to the marriage licence as a "hitting licence" (Strothman, 1985). The second phase is during the first pregnancy, and the third is after the first child is born. These three stages are the points at which major modifications in self-representation and increased narcissistic vulnerability take place (Strothman, 1985).

Wife battering is said to occur in 50% of marriages in the USA (Straus, 1978; quoted by Mason & Blankenship, 1987) and constitutes 26% of all police-reported violence in Scotland. Such violence may escalate, so that in excess of 25% of all murders in London are of wives killed by their husbands, and 44% of female murder victims are killed by husbands or lovers. Wife battering usually takes place in private – only about 2% of cases are reported, and charges are pressed in only a fraction of these cases. Nevertheless, the incidence of such violence is now on the increase, owing to greater reporting. The usual cause given for wife battering is the husband's need to maintain male dominance (Pahl, 1979), but a more detailed examination of this material shows that such husbands become violent when their sense of self-esteem is threatened, often by trivial actions such as the wife serving the children first, or the wife's reaction to being forced to adopt a humiliating role.

Such men often fall into the clinical category of narcissistic or borderline disorders, but the full range of personalities up to the apparently quite normal is encountered. "Thomas Corlett was a Civil Servant of impeccable character, but when his wife put a pot of German mustard and his favourite newspaper on the wrong side of the plate, his temper snapped and he killed her" (*The Times*, 10 July 1987). The altercation began as a battle of wills. In his evidence to the court, Mr Corlett related how he had placed the objects as usual on each side of the plate, when Mrs Corlett changed them over, shouting "That's where I want it and that is where I will put it." "She raised her hand and I thought she was going to hit me," he said. "I just grabbed her by the throat and we both fell to the floor. My mind then seemed to go blank." This case exemplifies how a formerly impeccable social self was breached when the omnipotent narcissistic demands in both partners tragically released an archaic imperative to silence and control the object upon whom he was dependent.

Mason & Blankenship (1987) found that abuse occurred more often in committed relationships. In husband battering, the type of women who were likely to inflict abuse were those who were highly stressed, had a high need for power and a low inhibition of activity. But when women struck out at men, they were usually struck in return.

Sharkey (1980) examined a group of child-abusing mothers and a group of controls for their sense of personal worth and self-esteem. Analysing the literature, Sharkey reported that child abuse in the USA was a major national problem, in spite of increased efforts at prevention and treatment. Low self-esteem was one of six characteristics of adults who abused children. The subjects were described as feeling worthless and devalued because of rejection and criticism during their childhood. These findings supported the role reversal hypothesis whereby abusive parents invest their children with the same negative feelings and critical attitudes which were displayed towards them by their parents. When these children became parents, they retaliated against their own children with the assaultive behaviour they experienced from their parents. Role reversal, with its underlying mechanisms of identification with the aggressor and turning passive into active, provides the unconscious setting and compulsion for violence against children.

Galdstone (1981) described the background of violence in child abuse. An atmosphere of discontinuity existed between the generations. There was a phobic preoccupation with danger and there were inadequacies in the assailants' capacity to handle their aggression.

As part of the ensuing cycle of deprivation and violence, such individuals suffer from ambivalence, usually on an oral-sadistic level. The lack of mastery over their aggressive drives leads to aggression being expressed purely as violence and not as mastery of the environment or creative activity, resulting in an empty personality with little pleasure in work. The next generation repeats the failure to master aggression.

Conclusion

The prevention of violence in future generations is best promoted by the encouragement of good parental rearing attitudes and practices. In particular these involve the maturation of narcissistic development and the maintenance of healthy sources of self-esteem, to assist in the diminution of the grandiose self and the formation of a more realistic ideal self, so that there would not be the necessity for recourse to violent body-ego expressions to shore up the cohesion of the self. Unfortunately the complexity of narcissistic development and the inherent aggressiveness in human personality ensure that violence is likely to be around for a long time.

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Controlled Comparison of Day-patient and Out-patient Treatment for Persistent Anxiety and Depression

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The effectiveness of day care versus out-patient care in the treatment of persistent severe anxiety and depression was compared in a controlled clinical trial. Of 96 consecutively referred patients meeting the entry criteria, 92 were followed up for six months. Patients were randomised to day care or out-patient care, and assessed at entry and at six months using the Standardised Psychiatric Interview and in terms of their time structuring and socialisation. Marked improvement in all three measures was seen for most of the day patients, but for few of the out-patients: this difference was highly significant for each measure. Day patients also rated themselves as coping more effectively and as more satisfied with their treatment. These differences could not be explained by differences in use of medication. Day treatment should remain an option for patients with persistent anxiety and depression resistant to out-patient treatment.

Given the current shift of emphasis from in-patient to community management in psychiatry, day hospitals are assuming an increasingly important role in service delivery. Their specific contribution, however, needs to be more clearly defined (Wilkinson, 1984; Vaughan, 1985). The literature comparing out-patient with day-patient treatment is less extensive than that comparing day-patient with in-patient treatment. In a randomised controlled British study, Tyrer & Remington (1979) compared out-patient with day-patient treatment for patients with diagnoses of anxiety, phobic or depressive neurosis. They found no important differences in clinical or social outcome and concluded that, "because out-patient care is more economic of time and personnel, it should be preferred to psychiatric day care for many neurotic disorders".

The current study also compares out-patient with day-patient treatment for anxiety and depression, but for a group not previously studied – patients referred for assessment for day treatment rather than routine out-patient referrals. We were interested to see whether it would be possible to demonstrate any benefit from attendance in this group with persistent symptoms resistant to out-patient treatment.

Method

All referrals to Dundee's only acute day hospital in the three years from mid-1984 to mid-1987 who met the following criteria were included in the study:

(a) predominant anxiety and/or depression in the absence of schizophrenia, mania, depression with delusions, organic brain disease, or alcohol or drug dependence

(b) symptoms of moderate severity, lasting continuously for a minimum of six months – patients were excluded from randomisation if they were assessed by the day-hospital consultant as being too well (requiring only out-patient support) or too ill (requiring more than out-patient support) or as having a specific requirement for day-hospital treatment (e.g. as part of a behavioural programme)

(c) willingness to accept day-hospital or out-patient treatment and co-operate in the study.

All patients gave informed consent and the study had approval from the ethical committee.

Initial interviews were carried out by the day-hospital consultant and follow-up interviews by a research senior registrar. The senior registrar was not made aware of the patients' treatment setting and made it clear at the start of her interview that it would be helpful if patients did not disclose what treatment they had received; it is unrealistic, however, to claim that single-blind conditions applied rigorously, as some patients spontaneously talked about their treatment. Day-hospital treatment with this patient group is usually for two to three months; we decided on a follow-up of six months from entry to assess progress a reasonable interval after day attendance.

In the initial interview patients were categorised according to the DSM–III criteria for anxiety and depression (American Psychiatric Association, 1980), although we departed from DSM–III in making separate ratings for anxiety and depression. The Standardised Psychiatric Interview (SPI; Goldberg *et al*, 1970), which gives an overall clinical severity score, was administered to patients initially and at follow-up. Patients were also asked about how they organised their activities and time (time structuring/organisation), and maintained their social contacts (socialisation) in a specially designed semistructured interview (Appendix). At follow-up patients completed a brief questionnaire on satisfaction with treatment and on coping.

The day hospital's main roles are community treatment of patients, (both neurotic and psychotic) who would otherwise require admission, and treatment of patients with severe persisting neurotic disability not responsive to or suitable for out-patient treatment alone. Attendance is generally from a few weeks up to several months, and there are up to 40 patients. Treatment is eclectic, with a focus on time structuring and socialisation, and a problem-orientated supportive/behavioural rather than psychodynamic approach. Staffing comprises three sessions per week of consultant time, three sessions per week of support medical time, three full-time trained nurses, and one full-time occupational therapist. Patients are referred by psychiatric colleagues, not general practitioners; colleagues are encouraged to refer patients to find out more about day treatment as one possible option rather than with the automatic expectation of attendance.

Patients allocated to continued out-patient treatment were seen approximately monthly and given advice on relaxation, anxiety management, and alternative approaches to time structuring and handling relationships. No constraints were put on use of medication in either setting. Limited input from psychologists, social workers, and community nurses was available to patients in both settings.

Patients were randomly allocated to day-hospital or out-patient treatment by opening a sealed envelope once the inclusion criteria had been met; they had, therefore, been assessed as suitable for both day-patient and out-patient treatment and had agreed to accept either. In practice the major difficulty lay in persuading those allocated to day treatment to attend, rather than in patients expecting day treatment being disappointed with out-patient treatment (perhaps not surprising in view of the substantial extra stress and time commitment involved in day attendance).

Of the 124 referrals who met the diagnostic criteria during the study period, all had had symptoms continuously for at least six months. Seven (mean PSI score 16.9) were excluded as too well, ten (mean PSI score 43.5) as too ill, and three (mean PSI score 32.3) because they required a specific behavioural programme: eight (mean PSI score 32.8) refused to accept day treatment as an option, but none refused out-patient treatment. The remaining 96 patients were randomised – 46 to day patient and 50 to out-patient treatment. Two out-patients but no day patients required in-patient care during the course of the study. All other patients completed at least one month of their assigned treatment. Two of each group were lost to follow-up.

The main analysis was of the 92 patients who completed follow-up to six months and was by intention to treat. The groups were comparable in initial characteristics (Table 1).

Statistical method

Differences between the groups for categorical variables were assessed by the χ^2 test, and Yates' correction was used for two-by-two tables. Comparison of clinical outcome was achieved by obtaining the difference between initial and final scores for each patient and comparing the two groups by the Mann–Whitney *U* test.

Table 1
Comparison of day and out-patients on entry

	Day patients ($n=44$)	Out-patients ($n=48$)
Age:		
<45	26	22
⩾45	18	26
Sex		
male	7	16
female	37	32
Marital status		
never married	7	4
married/cohabiting	24	27
separated/divorced	12	13
widowed	1	4
Employment		
working	6	5
not working	38	43
Diagnosis (DSM-III)		
depression		
major depressive	42	43
dysthymic	2	5
anxiety		
panic	17	21
phobic	9	8
generalised anxiety	16	15
no anxiety diagnosis	2	4
Alcohol use		
no problems	31	34
problems (but not dependency)	13	14
Interval since first contact		
two years or more	26	34
Most recent spell of adequate functioning		
within past year	7	4
not within past year	33	38
never as adult	4	6
Previously an in-patient		
yes	17	23
no	27	25

Results

The two groups were initially similar in their PSI scores (Fig. 1). After the six months of treatment a marked improvement was seen among most of the day patients, but among only a few of the out-patients. This difference was highly statistically significant (Mann–Whitney *U* test, $P<0.001$). The results were in the same direction for time structuring ($\chi^2=32.9$, d.f. $=2$, $P<0.001$) and socialisation ($\chi^2=23.0$, d.f. $=2$, $p<0.001$) (Table 2). On self-rating of coping, 36 out of 44 day patients rated themselves as coping effectively at least half the time, compared with 13 out of the 48 out-patients, a statistically significant difference ($\chi^2=28.9$, d.f. $=3$, $P<0.001$).

All patients were offered advice in relaxation and anxiety management and were advised of the drawbacks of longer-term use of benzodiazepines. With day patients this was accompanied by supervised relaxation sessions and individual and group discussion. Despite this, many patients

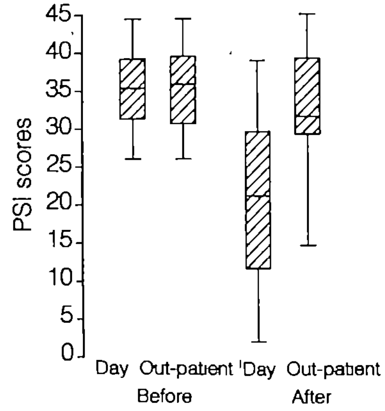

Fig. 1 PSI scores of patients randomised to day and out-patient treatment, before and after six months (highest and lowest ratings shown by bars, 25th–75th percentile by shaded box, median by horizontal line).

felt they continued to derive useful benefit from medication (although benzodiazepines were generally used in a variable-dose regime). There was little evidence of misuse or of escalating use. Patterns of drug use were similar in both groups before randomisation and at the end of follow-up (Table 3).

Of the 44 day patients, 37 were highly satisfied or satisfied with treatment compared with 28 of the 48 out-patients ($\chi^2 = 13.2$, d.f. $= 4$, $P < 0.05$).

The majority of patients in both groups were still in out-patient contact at six months. Mean duration of day treatment was 10.7 weeks (excluding two patients who were still attending at six months). There were no significant differences between the groups in mean number of contacts with a general practitioner (6.2 for day patients, 6 for out-patients), nor in the numbers seen by our community psychiatric nurse, social worker, or psychologist (only a small minority in each group).

Table 2
Time structuring and socialisation

	Worse	Same	Better
Time structuring			
day patients	0	6	38
out-patients	3	32	13
Socialisation			
day patients	1	11	32
out-patients	2	35	11

Table 3
Use of medication

	Day patients		Out-patients	
	Initally	At 6 months	Initially	At 6 months
Tranquillisers	11	16	14	13
Hypnotics	21	19	21	20
Antidepressants	20	18	28	26

Discussion

For these patients and treatment settings, day-hospital treatment was superior to out-patient treatment both clinically and on a number of other measures; the effect was not achieved by increased drug prescribing. This finding differs from previously reported work. Unlike the most comparable previous study (Tyrer & Remington, 1979), however, our patients had already been identified as potentially suitable for day treatment, and had more severe, long-standing illnesses. Treatment was in a small unit with an experienced team using a structured, problem-orientated approach; day and out-patient treatment differed in quantity of treatment and availability of peer-group support rather than in fundamental approach. Thus the results of this study complement rather than contradict previous work.

The possibility that deprivation of day treatment may have had an adverse effect on the out-patient group needs to be considered. However, as there was no evidence of deterioration in this group and as the majority had had symptoms for at least two years it seems unlikely that this factor had a significant part to play.

Is a reduction in clinical severity (as judged by PSI scores) of between a third and a half worthwhile? Objectively, it was accompanied by useful clinical improvement and this was confirmed by patients' self-rating of coping and by our assessment of how the patients used their time and socialised. On balance, therefore, the reduction in clinical score, although modest, was worthwhile.

Day-hospital treatment is more expensive than out-patient treatment. Even given the clinical benefit, can this expense be justified? The majority of patients had experienced continuous symptoms for at least two years and many for much longer. They suffered severe symptoms and major impairment of their day-to-day activities and relationships. The majority had had unsuccessful trials of antidepressants and had either been regarded as unsuitable for, had failed to engage in, or had had an unsuccessful attempt at, individual therapy (cognitive, behavioural or psychodynamic). In the authors' view these factors make the expense justifiable, although it would also be important to look at the possibility of reducing the amount of contact, if this is possible without detriment to clinical outcome.

Why did day patients improve? Although the study was not designed to answer this question, two possible contributory factors emerged from the data collected. Day-hospital attendance focused attention on time structuring and socialisation, and a clear improvement in these aspects of functioning may have contributed to clinical improvement rather than being secondary. Day-hospital staff also repeatedly advise and reinforce patients on how to cope with symptoms; the better coping ability of day patients at six months may reflect the effectiveness of this approach.

Although the extent to which it is possible to generalise from our results to settings outside Dundee is limited, our study does suggest a continuing place for day attendance as a second-line treatment for those patients who have failed to respond to out-patient treatment alone.

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Appendix

Time structuring and socialisation interview

Patients were asked about their routine, activities and social contacts over the preceding week. Open probes were followed by specific questions covering the aspects outlined below. The interview was concluded by obtaining a time budget for each weekend day and for one other day to check that the initial picture obtained was accurate.

Time structuring/organisation

Ask about basic routine (going to bed, getting up, feeding self), work, housework, conversation, reading, radio, music, television, hobbies, classes, sport, relaxation (sitting at peace). How much of the time was the patient actively engaged or comfortably relaxed? Did he/she require encouragement to maintain routine? Is he/she trying to rebuild routine? To what extend were activities disrupted by restless, unsettled behaviour?

0 = No problems with time structuring.
1 = Mild restlessness or inertia for minority of time. Limited disruption of usual activities, or activities maintained only with encouragement.
2 = Moderate restlessness or inertia up to half the time. Basic routine maintained. Moderate disruption of other activities, but regular attempts made to maintain routine and activities.
3 = Moderate restlessness or inertia for more than half the time, but no or infrequent severe restlessness. Some disruption of basic routine or basic routine maintained only with encouragement. Major disruption of other activities though some attempts still being made to maintain routine and activities.
4 = Regular acute disorganisation or continually in bed. Basic routine disrupted. Other activities dropped with little or no effort to restart them.

Socialisation

Ask about informal social interaction within the family, social contacts with extended family, neighbours and friends, and 'structured' social activities - church, bingo, social clubs; cover social aspects of sport and classes. How successfully was the patient maintaining day-to-day social interaction and other social activities? Did he/she require encouragement to interact and maintain social life?

0 = No problems with socialisation.
1 = Some unease, but only limited disruption of social contacts (or contacts maintained only with encouragement).
2 = Moderate disruption of socialisation, but still willing to maintain conversation/contact in familiar settings. Regularly attempting to keep up contacts.
3 = Most outside contacts avoided and some disruption of socialisation even in optimal settings. Some attempts still being made to maintain or restart socialisation.
4 = Interaction actively avoided despite encouragement, even with 'comfortable' friends or family.

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A Longitudinal Study of Psychological and Social Factors Affecting Recovery from Psychiatric Breakdowns

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Thirty-five people who had suffered a recent onset of symptoms of depression and/or anxiety were interviewed soon after being seen by a clinician, and again six months later. Symptom scores at the second interview were predicted by whether any plans subjects had formed by the first interview had gone wrong, by major non-health difficulties, and by internal, stable and global attributions made at the first interview about the event or difficulty that was most distressing before symptoms became severe.

There is now much evidence that psychological and social factors affect onset of depression (e.g. Brown & Harris, 1978; Brown *et al*, 1986; Oatley & Bolton, 1985). Much less is known, however, about factors that affect whether psychiatric symptoms become chronic.

We report here a longitudinal study of this problem with 35 patients. Many more would be necessary to examine with statistical precision the full range of factors that should be considered. Nevertheless, even with this small sample we can point to factors that are important in influencing the persistence of symptoms.

Our study was based on the following perspective. People have plans, aspirations or directions that inform their lives. They may cultivate these in areas such as work, sexual relationships, the family and leisure activities. Serious life events are typically, as argued by Oatley & Bolton (1985) and by Oatley (1988), events that remove a fundamental role from a person, and undermine a life plan in at least one such area. An onset of depression can then result if the person has no alternative means of maintaining a sense of himself or herself. Moreover, Perring *et al* (1988) have shown that conflicts between plans in different areas of life, for instance between work and family, are also associated with psychiatric symptoms.

According to this hypothesis, recovery from a depressive breakdown will be affected by how and when the person can construct new roles and new plans that are sustaining, and that do not involve substantial conflict.

The research reported here involved one interview shortly after the patient had seen a clinician for onset of symptoms, and a second six months later. We tested hypotheses about success of new plans and other psychosocial variables by predicting who would be symptomatic at the second interview. The study extends work of Tennant *et al* (1981), who found that events which neutralise threatening events allow remission of depression. It can be compared with work by Brown *et al* (1988), who have studied recovery from chronic depression in a community sample, and who also have surveyed the literature on recovery from depression. Our study is complementary to those in which severity of symptoms, chronic psychiatric problems, or constitutional factors predict recovery (e.g. Huxley *et al*, 1979; Mann *et al*, 1981; Murphy, 1983; Parker *et al*, 1985). It is also complementary to trials in which treatments such as pharmacotherapy and psychotherapy are evaluated.

Method

Thirty-five out-patients (23 women and 12 men) aged 18–65 (mean 38) took part. Criteria for entry into the study were that each had a recent onset or exacerbation of symptoms of depression and/or anxiety, without psychotic features, which had prompted him/her to seek professional help. People were included who had referred themselves or been referred for treatment. Any who had chronic psychiatric symptoms reaching at least borderline caseness as defined by Brown & Harris (1978) in the year before we saw them were excluded.

Patients were contacted through two agencies of each of the following three types: community mental health centres, psychology departments of psychiatric hospitals, and general practitioners' (GPs') surgeries. They were first asked by the clinician whom they were seeing if they would be willing to take part. Because of differing procedures at the different agencies, an overall response rate is approximate. Our most accurate figures come from our largest source, community mental health centres, where 64 people met our criteria and were approached: 27 agreed, 10 were not interviewed on the advice of their clinician, and 27 refused when asked by the clinician. This is a response rate of 42%. The GPs and hospital psychology departments were not able to give us accurate figures of how many people had been approached. Our follow-up rate, on the other hand, was very high. Only one subject of the original 36 interviewed at time 1 was not interviewed at time 2: he had moved and could not be traced. The original response rate was low because it took place in settings that were not research orientated, and especially because we ourselves were not able to ask people to participate but had to rely on clinicians who were not all motivated to ask people on our behalf. Although the low initial response rate may introduce some bias, our almost perfect follow-up rate allows conclusions to be drawn. We therefore concentrate our analyses not on general rates of recovery, but on factors predicting differences between people whose symptoms did and did not improve.

All who agreed to participate gave informed consent, and ethical permission was obtained from relevant local health authorities.

Procedure

Two semistructured interviews were conducted, the first at time 1, as soon as possible after being seen by a clinician, and the second at time 2, six months later. Except for one in a university office, all interviews were in participants' homes.

Symptom severity was assessed at both interviews using the shortened version of Present State Examination (PSE (9th edn); Wing *et al*, 1964), which includes 50 items for symptoms of depression, anxiety, phobic states and somatic disturbances. We constructed scores of all PSE symptoms of these types, counting 1 for each symptom of moderate intensity, and 2 for each of severe intensity. We defined time 0 as when the subject reported symptoms at their worst. At the first interview we assessed symptoms at time 0 and at time 1. The symptom score at time 2 was the outcome variable.

Predictive factors

We concentrated on five sets of predictive factors, as follows.

Plans and their outcome. Plans were assessed at time 1, partly from a schedule of activities in which participants were asked what they were doing in nine areas: occupation, domestic life, relationship(s), leisure, religion, self, children, finance, other; and asked which of these was the most important to them and whether other areas interfered with it. We then asked people to talk in an unstructured way about how they saw their next year, describing specific goals and plans, what obstacles they foresaw, and what problems they would have to solve. At time 2 we reminded them of each plan they had mentioned, and asked them to say what had happened in it, including conflicts and setbacks. The parts of the first and second interviews that concerned plans were tape recorded. From these recordings, summaries of plans were made for time 1 and time 2. Plans at time 1 were rated independently by each of us on a six-point scale of explicitness; ranging from 'steps being already taken' (coded 6) to 'no plans, hopes or fears' (coded 1). On each plan reported at time 1, we each independently rated whether the outcome at time 2 had matched aspirations expressed at time 1, or whether it had not worked out – a mismatch. Inter-rater reliability on explicitness, and on number of matches and mismatches was 0.68 or better. The ratings of the one of us who was blind to the subject's diagnosis were used in analyses.

Life events and non-health difficulties. We constructed a shortened version of Brown & Harris's (1978) Life Events and Difficulties Schedule asking during the first interview about events and difficulties occurring up to 38 weeks before time 0, when symptoms were at a maximum, and at the second interview for events and difficulties occurring between then and the second interview. Difficulties had to have lasted for at least four weeks, and we excluded any concerned with the participant's own psychiatric or physical health. Also, only events and difficulties rated as independent of the participant were considered. Our version of the schedule was based on Brugha *et al*'s (1985) list of the 12 categories of life events most important in long-term threat. All events and difficulties were rated independently for long-term threat by each of us, using Brown & Harris's methods, with one of us being blind to the participant's symptoms, and we counted those with moderate or marked threat. Inter-rater reliability was 89% for events, and 92% for difficulties. Discrepancies of rating between us on any event or difficulty (e.g. between moderate and low threat) were presented to Tirril Harris and the Bedford College team, and in such cases their ratings were used. The measures we used as predictor variables were number of severe events and number of major non-health difficulties at time 1.

Social support. All measures were those taken at time 1. Brown & Harris's measures of social support were used for marital relationships, for one close friendship and one close family member. In addition, we measured contacts with professional and paraprofessional resource people.

Coping. We augmented Parker & Brown's (1982) scale of six dimensions of coping behaviour by including three others, namely eating, watching TV, and doing something to take the mind off a problem. We asked separately about attempts to cope by solving the problem that had occurred, and coping with emotions, using at each interview a specific example of an event or difficulty that the participant had found distressing before that interview. Our measure was whether at time 1 each person used more problem-solving or more emotion-focused coping strategies.

Attribution. We used scales derived from Brewin (1986). At time 1 we asked participants to choose between two responses on internality of moral judgement (my responsibility – not my responsibility), internality as to social comparison (this would happen only to me – same would happen to others), stability (things like this always – never happen), and globality (things went wrong in every area of life – in just this one area); the first option in each set of parentheses scores high. We asked participants to apply these response options to the event or difficulty that had preceded their onset of symptoms which had been the most upsetting to them, and a score was made by summing responses to the four items.

Results

In this report we give three kinds of result. A fuller account is given by Perring (1987).

Firstly, for patients whose plans discussed at time 1 were all working out at time 2, we found that 13 had low PSE scores (below the median for all patients), whereas five had high PSE scores (above the median). By contrast, of those with at least one plan that was not working out at time 2, five had low and 12 high PSE scores. We excluded plans involving any apsect of psychiatric or physical health. This effect on PSE scores of a plan not working out was significant ($\chi^2=6.42$, d.f.1, $P<0.05$, approaching 0.01). There was also an association between number of plans at time 1 and number of mismatches at time 2 ($r=0.33$, not quite significant at the 5% level) but this indicates, rather obviously perhaps, that the more plans a person starts, the more likely it is that some will go wrong.

Table 1
Correlation coefficients (r) of predictor variables measured at time 1 with scores of PSE symptoms at time 1 and time 2

Predictor variable	With symptoms at time 1	With symptoms at time 2
No. of plans	−0.25	−0.23
Plan explicitness	−0.24	−0.14
No. of severe events	0.24	0.21
No. of major non-health difficulties	0.42**	0.35*
Social support		
all close others	−0.31	−0.30
marital	−0.30	−0.14
resource others	−0.27	−0.14
Coping		
use of problem-solving	0.17	−0.12
use of emotion-focused	0.14	−0.02
Attribution	0.25	0.50***

*$P<0.05$, **$P<0.01$; ***$P<0.001$, two-tailed, d.f. 33

Secondly, we performed correlations (r) between all predictor variables measured at time 1 and symptom scores measured at time 1 and time 2. These are given in Table 1, which shows that there was a non-significant association between the number of plans at time 1 and symptoms at time 1, indicating perhaps that more distress made it harder to make plans. The explicitness of plans at time 1 had non-significant correlations with symptom scores at time 1 and at time 2: less explicit plans were weakly associated with higher symptom scores. There were suggestions of associations between number of severe life events before time 0 and symptom scores at time 1 and time 2 but these were not significant, and when the Bedford College measure of caseness of depression was used as the outcome variable (with a three-point scale of non-case, borderline and case) the association was less strong. Major non-health difficulties at time 1 did have a significant association with symptom scores at time 2. There was a non-significant trend in the direction of more social support predicting fewer symptoms. Type of coping strategy at time 1 had no effect on symptom scores at either time 1 or time 2. The highest correlation in Table 1 was between attribution in relation to the most distressing event preceding time 0, and symptom score at time 2 – those who made internal, stable and global attributions at time 1 were significantly more likely to have higher symptoms scores at time 2.

Thirdly, we performed a regression analysis on symptom scores at time 2, entering into the equation the three most important control factors, namely symptom score at time 0, time since symptoms were at their maximum (i.e. time 2 – time 0) and social class (using the scale of Goldthorpe & Hope, 1974). These three variables together accounted for 20% of the variance. Other variables were then entered into the equation in stepwise fashion. Attribution scores and number of major non-health difficulties at time 1 were both significantly associated with symptoms at time 2 (P approximately 0.01 and 0.02 respectively). With this procedure, other variables (i.e. outcome and explicitness of plans, severe events, social support, and coping strategy) were not significant at the 5% level. Nor indeed was duration of psychological treatment, which we also entered into the equation.

Discussion

We have presented our findings in three parts. The first was about plans. Among factors implicated in whether symptoms became chronic was whether plans discussed soon after the breakdown were working out or not. Having at least one plan that had not worked out was significantly associated with symptoms remaining chronic at time 2.

The second aspect of our findings is shown in Table 1 – raw associations of predictor variables measured at time 1 with symptom scores at time 1 and time 2. The third aspect was a regression analysis with a specified order of entering variables. We present the regression analysis not with its conventional purpose of showing how much of the variance is contributed by each variable, but to check that when the most obvious general factors (i.e. symptom scores at time 0, duration of symptoms and social class) are controlled for, the largest associations in Table 1 remain.

Attribution, made shortly after symptoms had reached their maximum, was the most significant factor affecting chronicity. People who at time 1 made internal, stable and global judgements about the event they had found most distressing in provoking their symptoms, had more symptoms at time 2 than those who had made external, unstable and local attributions. The second most significant effect was of major non-health difficulties.

With our relatively small number of subjects, claims for significance of only a few factors can reasonably be made, and the regression procedure we have chosen is conservative in regard to some factors. It can be argued that since vulnerability factors, life events and difficulties largely explain the effect of social class on incidence of depression

(Brown & Harris, 1978), a regression analysis in which social class is controlled for may be biased against finding significant effects of social support, life events, or difficulties here. Table 1 indicates that there may indeed be a real association of both life events and lack of social support with symptoms scores at time 2. We believe, however, that it would require larger samples to establish whether these associations were significant.

Although the rate of recruiting into the study was low, this was because we depended on intermediaries to ask for participation. We cannot rule out the possibility that the clinicians who referred people to us preferentially selected those with higher motivation or self-confidence, although we believe the low initial response rate had more to do with the enthusiasm of referring clinicians for research and this would not have affected our sample so systematically. The results we describe, however, are of a longitudinal kind and the follow-up rate was very high, with all but one subject being interviewed at time 2. Hence there are good grounds for believing that factors which contribute generally to chronicity include: plans that do not work out; internal, stable and global attributions towards events seen as precipitating symptoms; and major difficulties.

Our study can usefully be compared with that of Brown *et al* (1988). They found, in a community sample of 48 women whose depression had lasted a year or more, that two kinds of happening affected recovery. One kind was a reduction in an ongoing difficulty, and the other was a fresh start of some kind, an event or decision that gave the woman some cause for hope that the future might become better. Whereas Brown *et al*'s study of recovery focused on people who had chronic symptoms which then improved, ours focused on factors that made symptoms of recent onset become chronic. Our results and those of Brown *et al* are thus complementary. In both studies chronic difficulties contributed significantly – we found that their presence was predictive of symptom scores remaining high after onset, and Brown *et al* reported that recovery was associated with reduction in the number or severity of difficulties. Similarly, both we and Brown *et al* found that new plans were important. We found that symptoms were more likely to become chronic if at least one plan conceived by time 1 had not worked out by time 2. Brown *et al* found that fresh starts were associated with recovery. In both studies, there were suggestions that here, as in the aetiology of onset, social support is important. We found a non-significant indication that lack of social support was important in maintaining symptoms at a high level. Brown *et al* found that the presence of support was significantly associated with shorter durations of chronic depressive symptoms.

The finding of ours which has no equivalent in Brown *et al*'s (1988) study concerns attribution. Attributional style, although much studied in quasi-experimental studies of mood deterioration, has not been shown to predict onset of clinically significant episodes of depression (see, for example, Brewin, 1985). It is therefore of all the more interest that a strong association of internal, global, and stable attributions about a distressing prior event or difficulty was associated with continuingly high levels of symptoms.

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Adversity in Groups with an Increased Risk of Minor Affective Disorder

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The hypothesis that the sociodemographic distribution of minor affective disorder can be explained by high-risk groups experiencing more psychosocial stress, defined in terms of life events and chronic difficulties, was tested. Linear logistic analysis of data from 275 subjects identified in a community psychiatric survey provided little support for this. Although high-risk groups uniformly experienced more psychosocial adversity, and adversity was strongly associated with disorder, this did not provide an adequate explanation for the increased risk of disorder This must therefore be explained in other ways.

In the last 30 years, there has been a growing literature on the social correlates of affective disorder. Connections have been sought at a macrosocial level, and it is clear that social variables can be used to identify high-risk groups (see e.g. reviews by Wing & Bebbington, 1985; Bebbington, 1988, 1991). It is now also generally accepted that life events bear a causal relationship to depressive illness (Paykel *et al*, 1969; Thompson & Hendrie, 1972; Cadoret *et al*, 1972; Markush & Favero, 1974; Myers *et al*, 1974; Brown & Harris, 1978; Cooke, 1981; Fava *et al*, 1981; Henderson *et al*, 1981; Costello, 1982; Surtees *et al*, 1986), and this relationship emerged strongly in our own data (Bebbington *et al*, 1981*a*). An obvious question follows: could the association of macrosocial variables with depression come about because they identify groups that experience frequent psychosocial adversity? This is the hypothesis examined here.

Consider, for example, the preponderance of women among sufferers from depression. Whether based on prevalence or on incidence studies, this finding is strikingly consistent (e.g. Weissman & Klerman, 1977; Rosenfield, 1980; von Zerssen & Weyerer, 1982), and applies equally to mild and severe depression (Adelstein *et al*, 1968; Pederson *et al*, 1972). Various explanations have been advanced. Although psychobiological influences may be important in a minority of cases, it is widely argued that psychosocial factors account for most of this difference between the sexes (Weissman & Klerman, 1977; Paykel & Rowan, 1979). For the experience of adversity to offer, on its own, an explanation of high rates of depression in women, it would have to be demonstrated that they experience more, or more upsetting, life events than men. This was indeed found by Brown & Birley (1968), but not by other workers (Uhlenhuth & Paykel 1973*a,b*; Uhlenhuth *et al*, 1974; Thoits, 1982). Women may however be exposed to more chronic problems than men (Radloff, 1975; Pearlin & Johnson, 1977; Pearlin & Lieberman, 1977).

Another sociodemographic association of affective disorder which could be explicable in terms of adversity is that with social class. In general, it is found that if a broad definition of depressive disorders is used, there is an inverse relationship between social prestige and disorder (e.g. Dohrenwend & Dohrenwend, 1969; Warheit *et al*, 1973; Uhlenhuth *et al*, 1974; Comstock & Helsing, 1976; Brown & Harris, 1978; Surtees *et al*, 1983). Persons of lower social class might be expected to experience more adversity (Faris & Dunham, 1939; Langner & Michael, 1963), and a number of studies confirm this (Dohrenwend & Dohrenwend, 1969; Dohrenwend, 1970, 1973; Myers *et al*, 1974; Pearlin & Lieberman, 1977; Brown & Harris, 1978; Kessler & Cleary, 1980; Bell *et al*, 1982; Thoits, 1982). This may explain their higher prevalence of disorder.

Other sociodemographic variables that are consistently associated with affective disorder include marital status and involvement in childcare (see e.g. Brown & Harris, 1978; Bebbington *et al*, 1981*b*; Bebbington, 1987, 1988; Der & Bebbington, 1987; Bebbington & Tansella, 1989). However, these variables operate in different directions on the risk of disorder for males and females. Being married and having care of young children appears to reduce risk for men, increase it for women. We certainly found this to be the case in the community psychiatric survey on which this paper is based (Bebbington *et al*, 1981*b*). Finally, we have recently explored the possibility that parity itself accounts for some of the gender difference in rates of affective disorder (Bebbington *et al*, 1990).

In the investigation reported here, we therefore tested the hypothesis that the associations of gender, social class and employment status with minor affective disorder arise as the consequence of an increased rate of psychosocial adversity in women, in the working class, and in the unemployed. Likewise, the differing relationships in the two sexes between affective disorder and marital status, parenthood and engagement in childcare can be explained by the distribution of psychosocial adversity.

Method

The methods and aims of this study have been described in detail elsewhere (Sturt *et al*, 1981; Bebbington *et al*, 1981*a*,*b*, 1984). It consisted of a two-stage survey of the general population of Camberwell in south-east London.

The two stages of the survey were based upon the PSE–ID–CATEGO system (Wing *et al*, 1974, 1978; Wing & Sturt, 1978). The Present State Examination (PSE) is a semi-structured interview schedule designed to elicit symptoms experienced in the last month. These symptoms then provide the input for the ID (Index of Definition) computer program, which allocates each subject to one of eight levels indicating the degree of confidence that sufficient symptoms are present to allow a clinical classification. Level 5 represents the threshold category, while subjects at levels 6, 7 and 8 are more definite cases. In our sample virtually all the cases found were of minor affective disorders (Wing *et al*, 1981), and we therefore examined the case group as a whole.

Trained interviewers from a reputable social survey agency interviewed a random sample of 393 men and 407 women aged 18–64. This sample had characteristics very close to those of the base population (Bebbington *et al*, 1981*b*). The interviewers used the 40-item version of the PSE and a questionnaire eliciting basic social facts about each subject, including gender, marital status, employment status, social class, and children. Social class was defined on the basis of the subject's occupation, using the 36 ranked classes of Goldthorpe & Hope (1974). We dichotomised these classes in the same way as Brown & Harris (1978), categories 1–22 being termed 'middle-class', and 23–36 'working class'. Employment was subdivided into full time (more than 30 hours per week) and part time. Marital status was categorised into 'single', 'married' and 'post-marital' (the divorced, widowed and separated). Subjects were also divided according to whether they had ever been parents ('parity') and whether they still had children under 15 living at home ('childcare').

We attempted to reinterview all those who were allocated to an ID level of 5 and above on the basis of the first interview, together with a random sample of non-cases. Of those who were classed cases from the agency interview, 82 were interviewed again. Of those defined as non-cases, 228 were also reinterviewed, making 310 in all.

The second interview was carried out four to six weeks later by the authors, and included the full 140-item PSE and a clinical history schedule designed to establish the onset and course of symptoms and the existence of prior episodes. The data from this second interview can be weighted back to the original sample to take account of the stratification involved in the second stage. The weighting procedure is described in detail by Bebbington *et al* (1981*b*).

Analyses in this paper are based on the 310 subjects comprising the second-stage sample, as it was then that life-event data were collected.

After the second PSE interview had been administered, we inquired about the symptoms that had been elicited earlier by the agency interviewers. Clear errors of rating on their part were corrected to give a 'best-bet' description of symptoms at the first interview. Corrections mostly involved down-rating.

In the analyses presented here, the *case group* comprises those who were deemed cases on the basis of the *corrected* first PSE, together with those who became cases between the interviews. The rationale for this decision is given by Bebbington *et al* (1981*a*). It seemed appropriate for the purposes of the current investigation to exclude chronic cases from analysis. This is because an excess of life events can only explain the increased case rate in sociodemographic high-risk groups if they precede the development of disorder. We therefore excluded cases developing more than a year before interview, as for them we recorded events before interview rather than before onset. This resulted in the removal from analysis of 35 subjects who were cases under the definition described above and whose condition was long standing. Analyses were therefore based on a maximum of 275 subjects, of whom 52 were acute cases as defined. In some analyses, numbers were slightly smaller because of missing data.

Life events were recorded and rated according to the procedure of Brown & Harris (1978). Events and difficulties elicited by each author were rated by the other two, who were blind to the subject's mental state and response to the event. The same four-point scale was used for rating the degree of threat the average person would see in the event: 1, marked threat; 2, moderate; 3, mild; and 4, no threat. The authors were trained by Brown and his colleagues and attained a high degree of reliability (Tennant *et al*, 1979).

Events were also divided into those that are 'logically independent' of disorder (usually because there is no way in which the subject's behaviour could have influenced them); those for which such an influence is improbable ('possibly independent'); and those that probably arose from behaviour consequent on psychiatric disorder ('probably dependent'). This division permits more weight to be placed on causal interpretations of relationships between events and disorders.

In the current paper we have restricted analyses to life events classed as 'independent' or 'possibly independent' and to those rated as being of 'marked' or 'moderate' threat. Although in theory 'possibly independent' events could have been occasioned by the development of psychiatric disorder, we chose to include them. We felt this provided a more realistic picture of the relationship without losing too much stringency. Events were included if they occurred in the three months preceding onset or, for non-cases, interview.

In compiling a history of chronic difficulties, the method of Brown & Harris (1978) was again followed. Chronic difficulties were elicited by using the same schedule as for life events and were rated in a similar way, except that a six-point scale was used to record severity. Difficulties in the top four levels of severity were included if they existed at interview, or, in cases, if they preceded onset. We chose this range of severity because we felt it was roughly equivalent to the severity of life events rated 1 and 2. In addition difficulties had to be rated as 'independent' or 'possibly independent'. Because of uncertainty of recall, we did not record difficulties that were not present at the time of interview. Separate analyses were carried out with life events alone and in conjunction with difficulties.

The findings reported here rely heavily on the use of linear logistic analysis. Full descriptions of this technique are available elsewhere (e.g. Cox, 1970). In essence it consists of the sequential addition of terms representing the effects of the independent variables on the dependent variable in such a way that the resulting models move closer to acceptable goodness of fit. The goodness of fit of a model is assessed by relating the scaled deviance (G^2) to a χ^2 distribution. Under the hypothesis that the model fits, G^2 approximately follows a χ^2 distribution with the same number of degrees of freedom as the model. The expected value of a χ^2 distribution is equal to its degrees of freedom, and a model can therefore be said to have reached acceptable fit when G^2 is roughly equal to this value. Up to the point when good fit, so defined, is reached, the statistical appropriateness of adding each additional term can be assessed by comparing the resulting changes in G^2 and in the degrees of freedom of the model with the distribution of χ^2. So, for example, a term that reduces the deviance of the model by 3.84 at the cost of a reduction of one degree of freedom can be said to contribute to the model, with a 5% probability of error.

Logistic analysis is a hierarchical procedure, as all lower-order terms must be incorporated before higher-order terms are added to the model. So, the inclusion of second-order effects of gender and adversity on disorder implies inclusion of the separate, first-order effects of gender and adversity on disorder. Within this limitation, the acceptable model is the most parsimonious with good fit.

The actual interpretation of the accepted model following log-linear analysis may require a return to the multi-dimensional contingency table on which it was based. The probable meaning of the significant interactions can then be determined.

In this paper, logistic analysis was carried out using the GLIM program (Payne, 1986). Although analyses were based on second-stage data, the figures were unweighted as this is the more statistically acceptable procedure.

Results

The first analysis is to establish whether the proposed hypothesis is worth testing for particular sociodemographic variables. This is done by seeing whether previously identified high-risk groups demonstrate high rates of adversity, whether defined by events alone or in conjunction with chronic difficulties. Table 1 indicates, somewhat surprisingly, that this is so for all the variables under examination. In consequence, it looks as if further analysis is justified in all cases.

Table 1
Life events and macrosocial variables

Macrosocial variable	% subjects with life event	% subjects with life event or difficulty	% subjects who are cases
Gender			
male	12.6	34.5	8.3
female	18.4	48.8	15.9
Employment			
full-time	14.9	38.9	9.3
part-time	22.2	53.3	8.6
not employed	25.0	62.5	20.4
Social class			
middle class	11.8	36.0	8.1
working class	23.5	51.5	16.7
Marital status			
males			
single	18.9	32.4	15.9
married	12.5	36.4	4.0
'post-marital'	20.0	60.0	32.3
females			
single	6.1	39.6	4.4
married	22.8	28.2	19.5
'post-marital'	35.0	60.0	22.8
Children at home			
males			
no	16.5	39.6	12.3
yes	10.3	28.2	0.0
females			
no	17.0	48.9	11.4
yes	27.5	52.9	24.7
Parity			
males			
non-parent	18.2	34.5	14.0
parent	12.0	37.3	4.6
females			
non-parent	12.0	38.0	8.8
parent	25.3	58.6	19.8

Values weighted to represent unstratified general-population sample. Only acute cases who were cases either on the corrected first interview or on the second interview are included. Chronic cases are excluded from the numerator and denominator. The prevalences thus differ from those quoted by Bebbington *et al* 1981(*a,b*)

The crucial question concerning the analyses that follow is this: does changing the order of adding the term linking the chosen sociodemographic variable with disorder to the model, so that it is introduced after the adversity–disorder term, lead to a significant reduction (or even the elimination) of its contribution to the model's fit? This would imply that associations with disorder can be accounted for in terms of associations with adversity.

The results are presented in some detail in Tables 2–7. The model of best fit is displayed, but in addition an indication is given in each case of the effect of introducing the uniformly powerful effect of adversity upon disorder *after* the interaction of the chosen sociodemographic variable with disorder.

Table 2 shows the results of testing the proposition that the association of gender with disorder can be explained by the fact that women experience more adversity. This is clearly not the case. There is, as expected, a large effect of adversity, but the model of best fit also requires the gender–disorder interaction. Adding the adversity–disorder term first does lead to a smaller contribution from the gender–disorder term, but not significantly so. Controlling for adversity defined as life events *and* difficulties actually reduced the change in the value of G^2 associated with the addition of the term linking gender and disorder below the conventional 5% level, but the shortfall is marginal ($G^2 = 3.69$, 1 d.f.).

Table 3 indicates that the adversity–disorder and employment-status–disorder terms are both required for the model of best fit although fit is improved by the latter only at the margins of significance. Reversing the order of incorporation of these terms makes no significant difference to the contribution of the employment-disorder term, implying that controlling for adversity does not really change the apparent tendency for the unemployed to suffer minor psychiatric disorders.

In our data, the influence of social class on disorder in any case fell short of statistical significance, so little can be made of the fact that its contribution virtually vanishes when we control for adversity, however defined (Table 4).

We now move on to more complex interactions. In Table 5, we display the model of best fit when the effects of gender, marital status and adversity on disorder are considered. In view of the hierarchical requirements of linear modelling, it is impermissible to enter the adversity–disorder term after the marital-status–gender–disorder term (this would involve adding a third-order term before all significant second-order terms had been incorporated). However, if this is done for purposes of illustration, the contribution of the latter is not significantly increased (the change in G^2 from adding in the term only increases by 2.56 and 1.50, for life events and overall adversity respectively). The tendency of marriage to have opposite effects on rates of disorder in the two sexes remains strong even after controlling for life events. When involvement in childcare is substituted for marital status as shown in Table 6, the model is very similar. The tendency for women with children at home to have high rates and men with children at home to have low rates of disorder therefore remains after controlling for adversity.

The overall findings of the study can therefore be summarised fairly simply. As might be guessed from Table 1, controlling for the relationship between adversity and disorder did tend to reduce the strength of interaction between disorder and the sociodemographic variables. However, in every case the reductions were small in comparison with the overall effects of these variables.

The subject of parity is explored in more detail in the following paper (this issue, pp. 40–45). The model of best fit did not require a parity–disorder term. Once more it is inadmissible to introduce the three-way interaction between parity, gender and disorder before the adversity–disorder term. This ordering of the model is therefore not shown in Table 7. However, if this is done for the purpose of illustration, the G^2 value of the contribution of the parity–gender–disorder term is increased to 3.77, which nears significance at the 5% level. There is thus little evidence from these data to support our initial hypothesis that interactions between sociodemographic variables and disorder arise from the frequency of adversity in high-risk groups.

Table 2
Logistic analysis of gender, adversity and disoder. model of best fit

	G^2	Change in G^2 with additional interaction
(a) Life events only		
Main effects	30.50 with 3 d.f.	
Plus adversity	4.75 with 2 d.f.	25.76 with 1 d.f.
Plus gender	0.57 with 1 d.f.	4.18 with 1 d.f.
or		
Plus gender	24.92 with 2 d.f	5.58 with 1 d.f.
Plus adversity	0.57 with 1 d.f.	24.35 with 1 d f.
(b) Life events and chronic difficulties		
Main effects	16.00 with 3 d.f.	
Plus adversity	3.70 with 2 d.f.	12.30 with 1 d.f.
Plus gender	0.01 with 1 d.f.	3.69 with 1 d f.
or		
Plus gender	10.42 with 2 d.f	5.58 with 1 d.f.
Plus adversity	0.01 with 1 d.f.	10.41 with 1 d f.

Table 3
Logistic analysis of employment status, adversity and disorder: model of best fit

	G^2	Change in G^2 with additional interaction
(a) Life events only		
Main effects	27.87 with 5 d.f.	
Plus adversity	6.98 with 4 d.f.	20.89 with 1 d.f.
Plus employment	2.36 with 2 d.f	4.62 with 2 d.f
or		
Plus employment	21.63 with 3 d.f.	6.24 with 2 d.f.
Plus adversity	2.36 with 2 d.f.	19.27 with 1 d.f.
(b) Life events and chronic difficulties		
Main effects	16.42 with 5 d.f.	
Plus adversity	6.19 with 4 d.f.	10.22 with 1 d.f.
Plus employment	1.77 with 2 d.f.	4.42 with 2 d.f
or		
Plus employment	10.18 with 3 d f	6.24 with 2 d.f.
Plus adversity	1.77 with 2 d.f	8.41 with 1 d f

The raw data for these analyses are presented in the Appendix.

Discussion

The general conclusion must be that although in some cases there are appreciable associations

Table 4
Logistic analysis of social class, adversity and disorder. model of best fit

	G^2	Change in G^2 with additional interaction
(a) Life events only		
Main effects	27.39 with 3 d f.	
Plus adversity	2.41 with 2 d f.	24 98 with 1 d.f.
Plus class	1.58 with 1 d.f.	0.83 with 1 d.f.
or		
Plus class	24.54 with 2 d f.	2.85 with 1 d.f.
Plus adversity	1.58 with 1 d.f	22.96 with 1 d.f.
(b) Life events and chronic difficulties		
Main effects	17 02 with 3 d.f.	
Plus adversity	4.57 with 2 d.f.	12 46 with 1 d.f.
Plus class	3.18 with 1 d.f.	1 39 with 1 d.f.
or		
Plus class	14.17 with 2 d.f.	2.85 with 1 d.f.
Plus adversity	3 18 with 1 d.f.	10.99 with 1 d f.

Table 5
Logistic analysis of marital status, gender, adversity and disorder: model of best fit

	G^2	Change in G^2 with additional interaction
(a) Life events only		
Main effects	42 67 with 11 d.f.	
Plus adversity	16.91 with 10 d f	25.76 with 1 d.f.
Plus gender	12.74 with 9 d.f.	4.18 with 1 d.f.
Plus marital status	12.20 with 7 d.f.	0.54 with 2 d.f.
Plus marital status × gender[1]	3.74 with 5 d.f.	8.46 with 2 d.f.
(b) Life events and chronic difficulties		
Main effects	28.91 with 11 d.f.	
Plus adversity	16.61 with 10 d.f.	12.30 with 1 d f.
Plus gender	12.92 with 9 d.f.	3.69 with 1 d.f.
Plus marital status	11.89 with 7 d f.	1.03 with 2 d.f.
Plus marital status × gender[2]	3 74 with 5 d f.	9.52 with 2 d.f.

[1] G^2 for the marital-status-gender–disorder term increases by 2.56 if it is introduced to the model before the adversity–disorder term.
[2] G^2 for the marital-status-gender-disorder term increases by 1 50 if it is introduced to the model before the adversity–disorder term

of macrosocial variables with adversity, there is little evidence that this greatly modifies the association of these variables with minor affective disorder. In other words, these associations must be mediated by factors other than adversity. So, for example, increased adversity would seem unlikely to provide a total explanation for the unemployment–disorder relationship, as unemployment is likely to have direct and detrimental effects on the subject's self-attitudes.

Table 6
Logistic analysis of childcare, gender, adversity and disorder. model of best fit

	G^2	Change in G^2 with additional interaction
(a) Life events only		
Main effects	46.70 with 7 d.f.	
Plus adversity	20 95 with 6 d.f.	25.76 with 1 d.f.
Plus gender	16.77 with 5 d f.	4.18 with 1 d f.
Plus childcare	16 76 with 4 d.f.	0.00 with 1 d.f.
Plus childcare × gender[1]	0.90 with 3 d.f	15.86 with 1 d.f
(b) Life events and chronic difficulties		
Main effects	35.16 with 7 d f	
Plus adversity	22.85 with 6 d f.	12.30 with 1 d f.
Plus gender	19.16 with 5 d.f.	3.69 with 1 d.f.
Plus childcare	19.10 with 4 d.f.	0.06 with 1 d.f.
Plus childcare × gender[2]	1.98 with 3 d.f.	17.12 with 1 d.f.

[1] G^2 for the childcare–gender–disorder term increases by 2 46 if it is introduced to the model before the adversity–disorder term.
[2] G^2 for the childcare-gender–disorder term increases by 1 22 if it is introduced to the model before the adversity–disorder term

Table 7
Logistic analysis of parity, gender, adversity and disorder: model of best fit

	G^2	Change in G^2 with additional interaction
(a) Life events only		
Main effects	34 68 with 7 d.f.	
Plus adversity	8.93 with 6 d.f.	25.76 with 1 d.f.
Plus gender	4.75 with 5 d.f.	4.18 with 1 d.f.
Plus parity	4.68 with 4 d.f.	0 08 with 1 d f not needed
Plus parity × gender[1]	2.77 with 3 d.f.	1 89 with 1 d.f. for best fit
(b) Life events and chronic difficulties		
Main effects	19.33 with 7 d.f.	
Plus adversity	7 03 with 6 d.f	12.30 with 1 d.f.
Plus gender	3.34 with 5 d.f.	3 69 with 1 d.f.
Plus parity	3.24 with 4 d.f.	0.10 with 1 d.f. not needed
Plus parity × gender[2]	0.23 with 3 d.f.	3 01 with 1 d f for best fit

[1] G^2 for the parity–gender–disorder term increases by 1 88 if it is introduced to the model before the adversity–disorder term
[2] G^2 for the parity-gender-disorder term increases by 0 77 if it is introduced to the model before the adversity–disorder term.

In this paper, we have chosen to analyse a range of macrosocial variables. There is likely to be some association between them: men of lower social class are somewhat more likely to be unemployed, for instance, and the overlap between being married, having children, and living with children is clear. These inter-relationships are important in themselves, and have been evaluated elsewhere (Bebbington *et al*, 1981*b*). However, they are of less relevance to the hypothesis under test in the current paper, where the focus is on the explanatory status of adversity.

We have conducted a considerable number of statistical tests in this paper, and significant interactions might have arisen by chance. This increases the weight that can be placed on our essentially negative findings.

There is very little in the literature that addresses the issues raised in this paper. With regard to gender, one possibility that has been suggested is that women respond badly to the life events they do experience, being more likely to develop depression as a result. This obviously implies some special susceptibility in women, which would require explanation. Two lines of evidence can be used to assess this. First, it appears that women do not assess events as being more stressful (Paykel *et al*, 1971). On the other hand, there is evidence of some increase in the relative risk and attributable risk of disorder in response to life events in women (Bebbington *et al*, 1981*a*; Cooke & Hole, 1983). This may be largely due to certain subgroups of women being especially prone to disorder in response to life events. If the index of attributable risk is used, the data both of Brown & Harris (1978) and Bebbington *et al* (1984) reveal that the risk of disorder attributable to life events was considerably increased in working-class women at home with young children.

It is also possible that women experience an excess of stress arising in ways that are not recordable as discrete life events, or even as chronic difficulties. Explanations of the sex differences in rates of depression have been conceived in terms of women's status in society and the roles available to them.

There was little effect of social class to explain: this may have led to a type-2 error of inference concerning the explanatory power of adversity. If it is yet the case that adversity itself can account for much of the social-class bias in rates of psychiatric disorder, this remains to be demonstrated. The question should probably be addressed through the analysis of findings more typical than our own in terms of the relationship between social class and disorder.

As we have seen, the literature provides some evidence of increased adversity in those of lower social class. However, this excess of adversity seems insufficient to account for class differences in risk of affective disorder. For example, Brown & Harris (1978) found approximately four times as much disorder in working-class than in middle-class women, and also showed that life events were somewhat commoner in the working-class group. However, within this group it was once more not those who were most likely to experience adversity who had the greatest probability of disorder. If the data both of Brown & Harris (1978) and of Bebbington *et al* (1986) are subjected to analysis based on the partitioning of contingency tables, class itself meets the requirements for a conditional effect on life events. In other words, class operates as a 'vulnerability factor': it is not so much the prevalence of adversity in the working class that accounts for their high rates of disorder as their susceptibility to the adversity they do experience. The concept of social class is shorthand for a whole range of differences of attitude, belief, and access to resources. The relationship between adversity, class and minor affective disorder is likely therefore to be more complicated than that implicit in the straightforward hypothesis tested in this paper.

Appendix

Gender – proportion of cases

(a) *Life events*	*Male*	*Female*
Life events	8/19	15/30
No life events	9/111	20/115
(b) *Adversity*		
Adversity	10/47	24/73
No adversity	7/83	11/72

Social class – proportion of cases

(a) *Life events*	*Middle class*	*Working class*
Life events	5/16	17/31
No life events	15/120	13/101
(b) *Adversity*		
Adversity	9/49	24/68
No adversity	11/87	6/64

Employment – proportion of cases

(a) *Life events*	*Full time*	*Part time*	*Non-employed*
Life events	10/25	1/6	9/14
No life events	16/152	4/25	9/47
(b) *Adversity*			
Adversity	14/61	3/20	14/34
No adversity	12/116	2/11	4/27

Gender and marital status – proportion of cases

(a) *Life events*	*Single*	*Married*	*Post-marital*
Male			
life events	3/7	4/11	1/1
no life events	5/30	3/77	1/4
Female			
life events	1/2	10/21	4/7
no life events	2/31	16/71	2/13

(b) *Adversity*			
Male			
adversity	4/12	4/32	2/3
no adversity	4/25	3/56	0/2
Female			
adversity	1/10	19/51	4/12
no adversity	2/23	7/41	2/8

Gender and childcare – proportion of cases

(a) *Life events*	*Not involved in childcare*	*Childcare*
Males		
life events	8/15	0/4
no life events	9/76	0/35
Females		
life events	7/16	8/14
no life events	10/78	10/37
(b) *Adversity*		
Males		
adversity	10/36	0/11
no adversity	7/55	0/28
Females		
adversity	10/46	14/27
no adversity	7/48	4/24

Gender and parity – proportion of cases

(a) *Life events*	*Non-parous*	*Parous*
Males		
life events	5/10	3/9
no life events	5/45	4/66
Females		
life events	4/6	11/24
no life events	5/44	15/71
(b) *Adversity*		
Males		
adversity	6/19	4/28
no adversity	4/36	3/47
Females		
adversity	5/19	19/54
no adversity	4/31	7/41

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Authors' details will be found at the end of the following paper, on page 45.

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Gender, Parity and the Prevalence of Minor Affective Disorder

P. E. BEBBINGTON, C. DEAN, G. DER, J. HURRY and C. TENNANT

Data from a two-stage survey of the general population were used to test the proposition that the over-representation of minor affective disorders among women was restricted to those who had had children, independently of current involvement in childcare. Initial analyses supported this proposition strongly. Subsequent linear logistic analyses were largely in favour of an effect of marriage rather than of parity, but it remains possible that part of the gender differences in rates of depression arises because of the effect of parity in raising female prevalence. If substantiated, the parity effect could operate through social or biological mechanisms; although its nature is unclear, it merits further investigation.

The over-representation of women among those who become depressed is perhaps the single most consistent finding in the epidemiology of affective disorder (Weissman & Klerman, 1977, 1985). The reasons for this have been the subject of considerable debate. The association of other demographic variables with gender and with disorder have always been taken to indicate that the explanation was likely to be social. In particular, the female preponderance of depression seems most marked among married subjects (Bebbington, 1987; Bebbington *et al*, 1981; Der & Bebbington, 1987; Gater *et al*, 1989).

The differences between the married and single seem unlikely at first sight to be of biological provenance. In an earlier analysis of data from the current study, we attributed high rates of depression in married women to involvement in childcare and the consequent restrictions on the opportunity for paid employment (Bebbington *et al*, 1981). However, Gater *et al* (1989) argued that the female preponderance in affective psychosis can be accounted for wholly in terms of parity: women who have never had children actually have a lower incidence of affective psychosis than men. The interpretation of this result must remain ambiguous, but it may give an important lead in elucidating the sex difference in the rate of depression. The current report is an attempt to replicate the effect of parity, but on a group largely suffering from minor conditions.

The central hypothesis therefore is that procreation increases the risk of psychiatric disorder for women, but not for men, such that the excess of psychiatric disorder in women is confined to those who have borne children. This effect of parity will be independent of whether the woman is still engaged in active childcare.

If the hypothesis is corroborated, it becomes important to search for possible contamination by variables that may influence the rate of psychiatric disorder and may also be related to parity. As in the paper by Gater *et al* (1989), the candidates considered are age and marital status.

Although the distribution of age of onset in minor affective disorders is different from that in affective psychosis, with a peak in young to middle adulthood (Bebbington, 1988), it would still seem necessary to control for age in a study of the effect of parity.

Method

The methods and aims of this study are as described in the previous paper (this issue, pp. 33–40). Trained interviewers interviewed a random population sample of 393 men and 407 women aged 18–64 years, using the 40-item version of the PSE and a questionnaire eliciting background information about each subject. The ID was applied to the PSE symptom profiles in order to determine 'cases' and 'non-cases'. A second interview was sought with all 82 'cases' and a random sample of 228 non-cases.

The second interview was carried out four to six weeks later by three of the authors (PB, CT, JH), and included the full 140-item PSE. The data from this interview can be weighted back to the original sample to take account of the stratification involved in the second stage.

Analyses in this paper are provided separately for each of the two stages of sampling. The problem with findings based on the first stage is that two of the eight agency interviewers did not rate as conservatively as the authors (Sturt *et al*, 1981). Because of this, a check was made during the second interview on the symptoms rated by the agency interviewers during the first. Symptoms were corrected only when there was evidence that: (a) facts had been misinterpreted (e.g. anxiety was rated in the absence of autonomic symptoms, or loss of weight due to dieting had been scored as loss of weight due to lack of appetite); or (b) the subject had on purpose or through inadvertence concealed information at the first interview. Specific symptoms such as depression and anxiety could be re-rated more confidently than non-specific ones like worry or nervous tension. There were however cases in which an obvious error had been made. This rating therefore represents an improvement over the agency PSEs, but still may err to an unknown extent towards too low a threshold. For this reason, findings based on the 'corrected' first interview should still be viewed less confidently than those from the second, although the first sample brings the advantage of larger numbers to the avoidance of type 2 errors of inference.

The basic sociodemographic description of our subjects was obtained at the first interview. In addition to age, gender, marital status and the existence of offspring, information was also obtained about primary responsibility for the care of children at home during the day. Subjects were rated as caring for children in this way only if there was at least one child at home during the day. This excluded children at school, at home on holiday, at home temporarily because of sickness, or attending pre-school for more than 15 hours a week. In principle, men could be rated as engaged in childcare in this sense; in practice, none was. Caring for such young children has been identified as an important correlate of the risk of psychiatric disorder in a number of studies (e.g. Brown & Harris, 1978; Surtees *et al*, 1983), including our own (Bebbington *et al*, 1984).

The findings reported here rely heavily on the use of linear logistic analysis described in the previous paper.

There is a strategic distinction between the traditional use of χ^2 to test for independence of the variables in a two-way table and using it to test for goodness of fit in a linear logistic model. In the first case, the aim is to *reject* the null hypothesis (that no association exists between the variables). Because we do not want to risk claiming a relationship where none exists, the hoped for result is a high value of χ^2 relative to the degrees of freedom. We therefore set the probability of type 1 error to a low value ($P=0.05$ or $P=0.01$). In contrast, in testing for goodness of fit, the hoped for result is the *acceptance* of the hypothesised model, and consequently a low value of G^2 relative to the degrees of freedom. In such a case the researcher is more concerned about type 2 error, that is, the rejection of relationships that exist in the population from which the sample was drawn. This may require trading off the risk of type 2 error against the dangers of type 1 error, by setting the probability of type 1 error at a relatively high value. This may mean accepting the contribution of an interaction term to the model despite a corresponding change in G^2 with a probability of less than the conventional 5%.

Logistic analysis is a hierarchical procedure insofar as all lower-order terms must be incorporated before higher-order terms are added to the model. So, the inclusion of second-order effects like that of gender/parity on disorder implies inclusion of all first-order effects involving those variables.

The actual interpretation of the accepted model following logistic analysis often requires a return to the multidimensional contingency table on which it was based. The precise meaning of the significant interactions can then be determined.

Logistic analysis was again carried out using the GLIM program, and analyses of the second-stage data were based on unweighted figures.

Results

The starting point of the analyses in this paper is shown in Table 1. The prevalence of cases in the second-stage sample has been weighted to be comparable with the first-stage sample and representative of the population sampled. Both the first- and second-stage samples show clearly that parity in women is associated with a considerable increase in the rate of psychiatric disorder, which is independent of having to care for children at home during the day. At first sight, therefore, it looks as though parity has an effect on disorder that persists after the hurly-burly of children's early years.

The effect of parity and gender on rates of minor affective disorder is illustrated by Table 2, summarising the logistic analysis carried out on both first- and second-stage samples. The results were similar. In neither analysis did the parity term on its own improve the fit of the model much, although in the second-stage sample the change in G^2 brought about by adding this term approached significance at the 5% level. The interaction of parity and gender is required to model the data from both stages, since when the separate effects of parity and gender are added, the model still deviates from acceptable goodness of fit. In reference to the second-stage data, we would not regard a model with a value of G^2 equalling 3.72 with one degree of freedom as being of adequate fit.

These results can be interpreted as follows. The effect of parity on disorder depends on the gender of the subject: it is associated with increased rates of disorder in women, but not in men. However, as will be seen, this effect does not wholly account for the influence of gender on disorder.

Table 1
Prevalence of minor affective disorder

	No. of subjects	% cases	Significance
First-stage sample			
Men without children	163	10.4	$\chi^2=2.4$, 1 d.f., NS
Men with children	230	5.7	
Women without children	141	11.3	$\chi^2=7.5$, 2 d.f., $P<0.02$
Women with children – engaged in childcare	52	25.0	
Women with children – not engaged in childcare	214	21.5	
Second-stage sample[1]			
Men without children	63	7.8	$\chi^2=0.1$, 1 d.f., NS
Men with children	84	5.1	
Women without children	53	5.8	$\chi^2=6.1$, 2 d.f., $P<0.05$
Women with children – engaged in childcare	23	20.9	
Women with children – not engaged in childcare	88	19.1	

[1] Data in the second-stage sample are weighted to take account of the stratification For this reason numbers are notional and differ from the actual numbers. However, the actual numbers have been used to calculate statistical significance

Table 2
Logistic analysis of gender, parity and disorder: model of best fit

	G^2	Change in G^2 with additional interactions
First-stage sample		
Grand mean	31.76 with 3 d.f.	
Plus gender	10.70 with 2 d.f.	21.06 with 1 d.f.
Plus parity	9.24 with 1 d.f.	1.46 with 1 d.f.
Second-stage sample		
Grand mean	16.20 with 3 d.f.	
Plus gender	6.73 with 2 d.f.	9.46 with 1 d.f.
Plus parity	3.72 with 1 d.f.	3.02 with 1 d.f.

In the nature of things, parous women as a group must be older than non-parous women, and the restrictive consequences of childcare persist beyond the time when the children are young enough to be at home in the day. Although in our original sociodemographic analysis age had little effect on rates of minor affective disorder (Bebbington *et al*, 1981), it would seem appropriate to control for this in our assessment of the effect of parity. We therefore carried out a logistic analysis based on a simple dichotomisation into those aged 39 or less and those aged 40 or over. These results are not presented in detail as the effect of age was not required for modelling the data from either stage, and the models of best fit were identical to those displayed in Table 2. Although there is no significant influence of age upon the relationship of parity on disorder, Figs 1 and 2 do suggest that the parity effect falls away in the oldest group of women (50–64 years).

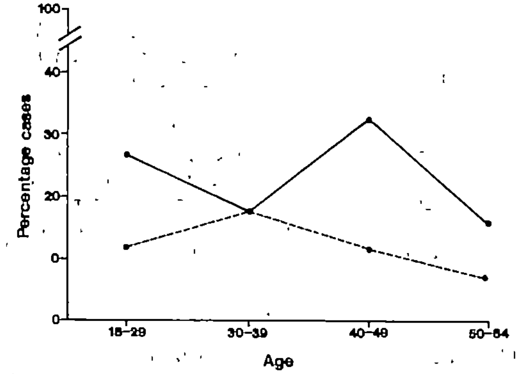

Fig. 1 Psychiatric disorder (ID ≥ 5) in women, by age (first-stage sample) (●–● non-parous; ●—● parous).

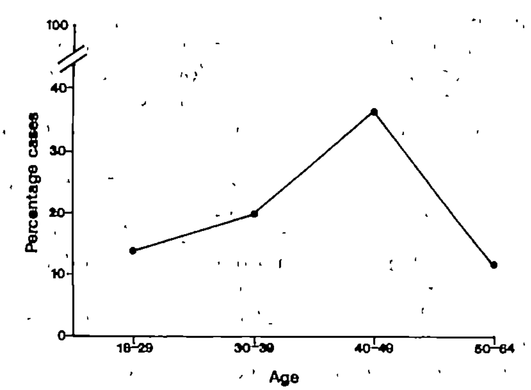

Fig. 2 Psychiatric disorder (ID ≥ 5) in parous women, by age (second-stage sample).

One way of simplifying the relationships of parity, gender and disorder is through the use of a composite term like that employed by Gater *et al* (1989). They created a new variable, 'SEXP', that divided their sample into men, women without children, and women with children. It is not completely justifiable to do this with the current sample as there are differences between parous and non-parous men, the latter actually having higher rates. However, for the sake of replication we carried out a logistic analysis of this type. In both samples, age again had no effect on disorder, and acceptable goodness of fit was obtained by using the SEXP–disorder interaction, which contributed to the model at beyond the $P < 0.001$ level of significance.

This result does add to the evidence of a gender–parity effect on disorder, but the construction of the composite term obscures how it might operate.

The final, and crucial, analysis concerns the potential confounding effect of marital status. In our original analyses, it appeared that this had a profound effect on the risk of disorder: although the divorced, widowed and separated of both sexes had high rates of disorder, comparison of the single and the married showed opposite effects in men and women. Married men had very low rates of disorder, and married women high rates. We attempted to explain this in terms of the influence of childcare and restrictions on employment for married women, but this explanation was not a complete one (Bebbington *et al*, 1981). Other sources of information support the idea that marriage protects women from affective disorder much less than it does men (Der & Bebbington, 1987; Bebbington, 1988).

Parity is overwhelmingly associated with marriage: is it parity or marriage that increases the risk of disorder in women? This question requires that the effect of parity on disorder be analysed together with that of marital status. This was done, again using logistic analysis, and the results are presented in Tables 3 to 5. (Table 3 gives the actual data from the two samples.) The analysis was conducted in two ways. In the first, the single were compared with the married; in theory this might allow conclusions about whether it is the married or the parous state that raises risk. However, these states are so intimately related that an alternative strategy is needed to separate them. The second analysis therefore compares the married with the 'post-marital', that is, the divorced, widowed and separated; this comparison is more likely to permit statements about the independent effect of parity, because the post-marital are as likely as the married to have had children.

We first compare the single with the married (Table 4). The pattern of results from the first- and second-stage samples is not very different, although the principle of parsimony actually yields different models of best fit. In both samples, gender is strongly related to psychiatric disorder, whereas parity and marital status have no significant overall effect. However, second-order interactions improve the goodness of fit significantly. In the first-stage sample, the most parsimonious model is based on parity and incorporates the second-order interaction of gender and parity. When this is done, no other second-order interaction adds significantly to the model. However, a model based on marital status and including the second-order interaction of gender and marital status on disorder is only marginally of worse fit.

Table 3
Cases of minor affective disorder as proportion of total subgroups (raw data used in Tables 4 and 5)

	Single		Married		'Post-marital'	
First-stage sample						
Male						
parent	0/2	(0%)	10/214	(5%)	3/14	(21%)
non-parent	12/105	(11%)	3/52	(6%)	2/6	(33%)
Female[1]						
parent	1/10	(10%)	44/213	(21%)	13/42	(31%)
non-parent	8/85	(9%)	5/44	(11%)	3/12	(25%)
Second-stage sample						
Male						
parent	0/1	(0%)	4/73	(5%)	3/5	(60%)
non-parent	5/41	(12%)	1/19	(5%)	1/3	(33%)
Female						
parent	0/4	(0%)	27/93	(29%)	6/20	(30%)
non-parent	3/31	(10%)	2/15	(13%)	1/7	(14%)

[1] Marital status was unknown for one woman from the first sample.

Table 4
Logistic analysis of gender, parity, marital status (single v. married, post-marital excluded) and disorder: model of best fit

	G^2	Change in G^2 with additional interactions
First-stage sample		
Grand mean	29.84 with 7 d.f.	
Plus gender	12.41 with 6 d.f.	17.43 with 1 d.f.
Plus parity	11.56 with 5 d.f	0.85 with 1 d.f
Plus gender × parity	2.49 with 4 d.f.	9.06 with 1 d.f.
or		
Plus marital status	12.17 with 5 d.f.	0.24 with 1 d f
Plus gender × marital status	2.85 with 4 d.f.	9.33 with 1 d.f.
Second-stage sample		
Grand mean	22 94 with 7 d.f.	
Plus gender	10.39 with 6 d.f.	12.55 with 1 d.f.
Plus marital status	8 99 with 5 d.f.	1.41 with 1 d.f
Plus gender × marital status	2.85 with 4 d.f.	6.13 with 1 d.f.
or		
Plus parity	8.48 with 5 d.f.	1.91 with 1 d.f.
Plus gender × parity	3.69 with 4 d.f.	4.79 with 1 d.f.

Table 5
Logistic analysis of gender, parity, marital status (married v. post-marital, single excluded) and disorder: model of best fit

	G^2	Change in G^2 with additional interactions
First-stage sample		
Grand mean	41.88 with 7 d.f.	
Plus gender	13.72 with 6 d.f.	28 15 with 1 d.f.
Plus marital status	6.17 with 5 d f.	7.55 with 1 d.f.
Plus gender × marital status	2.83 with 4 d.f.	3.34 with 1 d.f.
Second-stage sample		
Grand mean	26 13 with 7 d.f.	
Plus gender	13.68 with 6 d.f.	12.45 with 1 d.f.
Plus marital status	11 83 with 5 d f.	1 85 with 1 d.f.
Plus gender × marital status	3.10 with 4 d.f.	8.73 with 1 d.f.

This marginal difference between alternative models is repeated in the second-stage subsample. Once more, the first-order effects of parity and marital status add little to that of gender. Second-order terms are needed for adequate fit, and significantly improve the model. However, this time the model with slightly better fit is based on marital status and incorporates the interaction of gender and marital status on disorder. These results emphasise the difficulty of separating the effects of marital status from those of parity.

When we move on to compare the married with the post-marital, the model of best fit in the first sample probably involves a second-order interaction of gender and marital status on disorder, as without this term the G^2 value for the model is still 6.17, with five degrees of freedom. However, despite being needed for acceptable fit, the addition of this term only reduces G^2 by 3.34, which falls short of significance at the 5% level. In the second-stage sample, the contribution of the gender–marital-status second-order interaction was more clearly necessary for the model of best fit. In neither sample was parity needed, either alone or in interaction with gender, and its effect was small.

Discussion

The study here differs in important respects from that of Gater *et al* (1989). Our subjects suffered predominantly from fairly minor depressive and anxiety states and few had seen psychiatrists. Social rather than biological factors might therefore be expected to play a part in aetiology. Gater *et al*, in contrast, studied first admissions with affective psychosis, and thus incidence, whereas we studied prevalence.

Nevertheless, there is *prima facie* evidence from our data that parity increases the prevalence of minor affective disorder in women, and that this effect does not depend on the obligations of full-time childcare. Moreover, while not accounting for it wholly, the effect makes a significant contribution to the sex difference in rates of disorder. This effect remains when the effect of age is controlled for.

However, the effect of parity does not stand out beyond that of marital status; in other words, a statistically more acceptable account is provided by saying that married women have high rates of disorder, rather than that parous women have high rates. An effect of parity cannot be entirely ruled out, although its significance would remain unclear, as it could indicate adverse social consequences or biological changes, or both. It may also reflect different things at different ages, and in different types of affective disorder.

One social explanation for the effect of parity or of marital status that we were able to explore was the possibility that parous and married women experience more psychosocial adversity (in the form of life events or chronic difficulties) than their single or nulliparous sisters. We report this possibility in the previous paper (this issue, pp. 33–40). We ran the logistic analyses using measures of life events, on their own and in conjunction with chronic difficulties. Analyses were run twice, once for marital status and once for parity. The inclusion of adversity did not eliminate the need for a second-order interaction between marital status, gender, and disorder. In other words, for women there is something about the married role that is associated with psychiatric disorder independently of the possibility that married women may experience more measurable adversity than their single counterparts. The results for parity were different: parity was not required for the model of best fit, implying that its effect can be explained in terms of parous women having more life events.

In our view, the central proposition that parous women have increased rates of affective disorder remains worthy of further investigation. This is particularly so in view of the mounting evidence about the effect of marriage on the risks of affective disorder for women. Many researchers will have data readily available for testing the effect of parity, and we encourage them to make the appropriate analyses. This may indicate the need for specific studies to determine the mechanism of the effect.

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The Relationship between Post-natal Depression and Mother–Child Interaction

ALAN STEIN, DENNIS H. GATH, JANET BUCHER, ALISON BOND, ANN DAY and PETER J. COOPER

The study was based on an index group of 49 mothers who had had depressive disorders in the post-natal year, and 49 control mothers who had been free from any psychiatric disorder since delivery. Nineteen months after childbirth, the interaction between mother and child was assessed by blind assessors using defined observational methods. Compared with controls, index mother–child pairs showed a reduced quality of interaction (e.g. mothers showed less facilitation of their children, children showed less affective sharing and less initial sociability with a stranger). Similar but reduced effects were seen in a subgroup of index mothers and children where the mother had recovered from depression by 19 months. Social and marital difficulties were associated with reduced quality of mother–child interaction.

There is good evidence for a link between depressive disorder in mothers and emotional disturbance in their children (Richman *et al*, 1982; Billings & Moos, 1983; Pound *et al*, 1985). The reasons for this link are not established, but Rutter & Quinton (1984) have suggested several possibilities, including the influence of the mothers' depression on the way the mother and child interact.

Two studies have thrown light on such interaction. Firstly, in a study of children aged two to three years, Radke-Yarrow *et al* (1985) used Ainsworth's Strange Situation and found that insecure attachment was more frequent in children whose mothers had a history of major depression than in those whose mothers had no such history. The evidence for insecure attachment was the child's avoidance of and resistance to the mother when reunited with her after brief separation. The second study also examined children aged two to three years (Cox *et al*, 1987). Compared with children of non-depressed mothers, those of depressed mothers showed more behaviour problems and more difficulties in expressive language. The important findings were that depressed mothers were less likely to use explanations, suggestions and questions when dealing with their children, and more likely to ignore their children's requests. Depressed mothers were also less likely to pick up cues from their children; if they did so, their responses were less likely to be taken up by the children. The complexity of possible causal factors was shown by the finding that depressed mothers were also having more marital and social difficulties.

When reporting their study of children aged two to three years, Radke-Yarrow *et al* (1985) commented that: "the depressed parent is the primary environment of the young child . . . the behaviours and mental status of a depressed person are all potentially interfering with the functions and responsibilities of a care giver and with the development of a good affective relationship with the child". It is arguable that younger children (up to the age of 18 months) are likely to be particularly vulnerable to effects of this kind, since they are entirely dependent on their carers. Hence studies of mothers who were depressed in the post-natal year should throw special light on mother–child interactions.

Field (1984) found that the young infants of post-natally depressed mothers (compared with those of non-depressed mothers) showed less frequent positive and more frequent negative facial expressions. They also vocalised more. The depressed mothers showed similar differences in facial expressions; they vocalised less and spent less time looking at and touching their children. In a recent study, Murray (personal communication) found that post-natally depressed mothers were slower to respond to their children; the latter looked at their mothers less and showed more self-directed activity.

These studies have largely focused on mothers with concurrent depression. It is arguable however that mother–child interactions should also be studied after the mother has recovered from her depression. Post-natal depression provides a good opportunity for studying this question since it is often of relatively short duration. Thus, by studying the mothers and children during the second year of the children's lives, a good proportion of mothers will have recovered from their depression while some will have remained depressed. We therefore examined mother–child interactions in three groups 19 months after the child was born. In the first group the mother was depressed during the post-natal year and also at 19 months; in the second, the mother was depressed in the post-natal year but not at 19 months; and in the third the mother had been free from depression throughout. The aim was to discover whether impaired mother–child interaction was more frequent in (a) mothers who had been depressed during the post-natal year when compared with those free from depression; (b) mothers who had recovered from their depression by 19 months when compared with mothers free from depression (since the birth of their children).

Method

In a previous study, Cooper *et al* (1988) had examined a large series of 483 mothers four times: two months before childbirth, and three, six and 12 months after delivery. At each assessment the mothers had completed the 60-item General Health Questionnaire (GHQ; Goldberg, 1972). Mothers with a GHQ total score of 12 or above had been further examined with a standardised psychiatric interview, the Present State Examination (PSE; Wing *et al*, 1974); the depression rating scale (MADRS) devised by Montgomery & Asberg (1979) was also completed. Sixty women had been identified as PSE cases on at least one of the three post-natal assessments.

In the present study, 19 months after childbirth 49 of these 60 women were assessed with their infants. Eleven of the 60 were omitted; of these, two had had twins, two were known to the research assessors, and the rest had moved away. Also assessed was a control group of 49 mothers who had scored below 12 on the GHQ at all three post-partum assessments; they were individually matched by age, social class and parity. Their sociodemographic characteristics are given in Table 1. Fortuitously equal numbers of mothers in both groups were cohabiting with male partners.

Table 1
Sociodemographic characteristics of mothers

	Index group (n=49)	Control group (n=49)
Median age (range): years	27 (16–40)	27 (17–37)
No. in middle class	23	22
No. married and/or stably cohabiting	45	46
No. primiparous	14	14

Assessments

Index and control mothers were examined on two occasions when the child was 19 months old. Firstly, an independent research assessor examined each mother's mental state with the PSE and the MADRS. The assessor confirmed that none of the control mothers had had any psychological difficulties since delivery. She also took the opportunity to assess recent life events (Paykel, 1980) and chronic social difficulties. This interview ascertains the occurrence of potentially stressful events during the previous 12 months under six headings: work, finance, health, relationships, legal and other. The interview was modified and extended to make a detailed assessment of any chronic social difficulties, for example marital and housing problems. Ratings of 'objective negative impact' and 'the degree of independence' for each life event was made by the interviewers (as recommended by Paykel & Mangen, 1980, unpublished manual). Chronic social difficulties were scored on a five-point scale from absent to severe. Inter-rater reliability for ratings of events and difficulties was found to be satisfactory (weighted kappa 0.76–0.94).

Secondly, within the following week, each mother and child were assessed by one of two research psychiatrists, who were blind to the mother's index/control status. Five assessments were made in the following order.

Child's sociability to a stranger

This assessment was based on a semi-standardised procedure devised by Stevenson & Lamb (1979) to assess the child's initial responses to a stranger in the mother's presence. In this procedure, which was originally designed for laboratory use, the stranger makes a series of increasingly intrusive approaches to the child. In the present study, the technique was first practised in the laboratory and later adapted for the home. To adapt the procedure for use in the home, some elements of Thompson & Lamb's method were omitted, but the essentials were the same as theirs.

The test lasted three to five minutes. It began with the child sitting on the mother's lap. The stranger (the research psychiatrist) offered the child a toy, and then tried to initiate a 'give-and-take' exchange of the toy. Next an assessment was made of the child's response to being placed on the floor. Finally, the researcher moved to the floor, and again offered the child a toy, and again tried to start a 'give-and-take' exchange. The child's response to each initiative was rated on a 1–5 scale ranging from withdrawn/distressed to utgoing/friendly (see Appendix for details). The interrater reliability for this assessment was 0.78 (weighted kappa).

Mother and child in structured play

The mother and child were observed at play in three situations each lasting three minutes. The mother was asked to behave as she usually would.

Firstly, the child was given a large plastic nut and bolt; this was chosen as a simple toy which was likely to interest the child only briefly and would require substantial effort by the mother to sustain the child's interest. Secondly, the child was given a toy hippopotamus which could be pulled on a string; also five plastic shapes could be posted through the animal's back and retrieved from its mouth. This was chosen as a more complex toy, which was likely to interest but also to frustrate the child if the mother did not help. Thirdly, the child was given a picture book and the mother was asked to read it with the child.

These situations were chosen in order to assess how the mother and child dealt with them; for example, how the child approached the toy, and how the mother maintained the child's interest and dealt with any difficulties. For this purpose six categories of behaviour were defined:

(a) child approaches assessor with/without toy
(b) child plays with toy without involving mother
(c) child and mother interact verbally or non-verbally in playing with toy
(d) mother ignores child
(e) mother facilitates child (this fifth category, derived from the work of Mills *et al* (1985), was defined as any maternal behaviour (verbal or non-verbal) which

Table 2

Structured play results for (a) 49 cases v. 49 controls, (b) 25 recovered cases v. 25 controls, and (c) 14 index subjects with severe chronic social difficulties (SCSD) v. 35 index subjects without SCSD

Structured play items	(a)			(b)		(c)		
	Controls ($n=49$)	Cases ($n=49$)		Cases ($n=25$)		SCSD ($n=14$)	Non-SCSD ($n=35$)	
	Mean	Mean	Z	Mean	Z	Mean	Mean	Z
Child plays alone	3.1	3.6	0.79	3.4	0.547	3.6	3.6	0.47
Child turns to observer	0.3	0.5	1.3	0.2	0.59	0.6	0.5	0.9
Child's affective sharing	3.8	2.4	2.9***	2.5	2.24**	1.1	3.0	2.1**
Mother and child interact	21.1	17.0	2.7***	17.7	1.6	14.1	18.3	1.5
Mother ignores child	0.8	1.5	1.16	1.6	1.34	2.4	1.1	1.74
Mother facilitates	20.9	15.8	2.78***	17.1	1.91*	10.8	17.8	2.4**

*$P<0.06$, **$P<0.05$, ***$P<0.01$

helped to maintain the child's interest and attention in playing with the toy; for example, the mother might rattle the hippopotamus and open its mouth to indicate how the shapes could be retrieved.)

(f) child shows affective sharing (this type of behaviour was defined as the child "smiling and vocalising and/or smiling and showing toys during joint play" (Waters *et al*, 1979); for example, the child undoes the nut and bolt, then looks at the mother, smiles, and shows her the toy.)

Ratings of these six categories were made for six 30-second periods during each three-minute period. The ratings were: absent, 0; present once or twice, 1; present repeatedly or persistently, 2.

Developmental assessment of child

The Denver developmental screening test (Frankenberg & Dodds, 1967) was used. It is made up of four subscales: (a) gross motor; (b) language; (c) fine motor-adaptive; (d) personal and social.

Child's response to mother's departure and return

The mother was asked to leave the room and the researcher assessed the child's resultant distress on a four-point scale. The mother returned a minute later, and an assessment was made of the child's acknowledgement of her and approaches to her.

Global ratings of mother and child

At the end of the interview, the assessor made global ratings of the mother's behaviour, the child's behaviour, and the interactions between mother and child. The mother was rated for intrusiveness (cuts across or disrupts flow of child's play, takes over and even excludes), warmth (extent to which she uses endearment and physical contact) and rapport (ease of communication, reflected in her ability to talk and listen to the child). The child was rated for concentration (maintaining interest and sustaining attention to task in hand) and negative responses (any including crying, tantrums, sulks and anger). Each item was rated on a 1–7 scale (mode 4, 1 = very low, 7 = very high).

Reliability

Inter-rater reliability checks were made regularly during the study with the following results: (a) for time-sampled ratings and measures of child's reaction to mother's departure and return: range 0.77–0.92 (weighted kappa); and (b) for global measures: range 0.62–0.82 (weighted kappa).

Results

As mentioned under Method, 49 index mothers had previously been identified as PSE cases in the 12 months after childbirth (Cooper *et al*, 1988). On allocation to PSE CATEGO classes, 39 were classified as having depression, and ten as having anxiety. Of the ten anxiety cases, seven could be deemed to have suffered from significant depression because they had positive ratings for 'depressed mood' on the PSE and/or scores over 18 on the MADRS. Of these seven women, three scored over 18 on the MADRS and on the PSE symptom 'depressed mood', three scored over 18 on the MADRS only and one scored on the PSE symptom 'depressed mood' only. These seven women were combined with the 39 PSE depressives into a group of 46 'previously depressed' index mothers.

In the present study, at the 19-month follow-up, nine of these 49 original PSE cases were still found to be PSE cases; all nine had been previously classified as having depression. Among the 40 women who had become non-cases at follow-up, 15 were at levels 3 or 4 (i.e. still with significant symptoms) on the PSE Index of Definition, while 25 were at levels 1 or 2.

Statistical analyses

The statistical techniques used included Wilcoxon tests for matched pairs, Mann–Whitney U-tests, χ^2 and Fisher exact probability tests and Spearman's rank correlations as appropriate.

The 49 index and 49 control mother–child pairs were first compared. With regard to structured play (Table 2), in the index group the children showed significantly less affective sharing ($P<0.01$); the mothers showed significantly less facilitation ($P<0.01$); while the mothers and children showed significantly less interaction ($P<0.01$). No significant differences were found on the measures of

Table 3
Global ratings for (a) 49 cases v. 49 controls, (b) 25 recovered cases v. 25 controls and (c) 14 index subjects with SCSD v. 35 index subjects without SCSD

Global ratings	(a) Controls ($n=49$)	(a) Cases ($n=49$)	(a)	(b) Cases ($n=25$)	(b)	(c) SCSD ($n=14$)	(c) Non-SCSD ($n=35$)	(c)
	Mean	Mean	Z	Mean	Z	Mean	Mean	Z
Maternal intrusiveness	3.2	3.2	0.4	3.4	0.76	2.9	3.3	1.75
Maternal warmth	4.8	5.0	0.48	5.0	0.06	4.2	5.3	2.29**
Maternal rapport	5.2	4.4	2.4**	4.4	1.9*	3.6	4.7	1.95**
Child concentration	4.7	3.7	2.8***	3.7	2.1**	3.3	3.8	1.0
Child negative responses	3.4	4.1	2.38**	3.6	1.3	4.6	3.9	1.2

*$P<0.06$, **$P<0.05$, ***$P<0.01$

child playing alone, child turning to observer, or mother ignoring child. With regard to global ratings (Table 3) index children showed significantly less concentration ($P<0.01$), and significantly more negative responses ($P<0.05$) than control children. Index mothers showed significantly less rapport ($P<0.05$) than control mothers, but did not differ significantly in warmth or intrusiveness. With regard to the other three assessments, there were also some significant differences. When their mother left the room, 14 index children and 4 control children showed marked distress ($\chi^2=5.5$; d.f. $=1$; $P<0.05$), although when their mothers returned, these two groups of children did not differ significantly in their responses. Significantly less sociability with a stranger was shown by the index children than by the control children ($Z=3.4$; $P<0.01$). Of the numerous items on the Denver development assessment, only one significant difference was found between the groups: 19 index children as against 30 controls were able to combine two words ($\chi^2=4.1$; d.f. $=1$; $P<0.05$).

All the analyses reported above for the 49 index women were repeated for the subgroup of 46 previously depressed women (excluding anxiety cases). The only notable difference was that on the language item combining two words on the Denver development scale, the index v. control difference, which was significant for the 49 index patients, was not significant for the subgroup of 46 when compared with their controls.

A comparison was also made between two groups of index mothers: (a) nine who were PSE cases in the post-partum year, and again at 19-month follow-up; and (b) 25 who were PSE cases in the post-partum year, but who had not recovered by 19 months. No significant differences were found, possibly because of the small number of PSE cases at 19 months. However, there was a trend for the children of mothers who were still PSE cases at 19 months to show less affective sharing ($Z=1.2$; $P<0.2$), less sociability ($Z=1.74$; $P<0.08$), and greater distress when the mother left the room ($\chi^2=1.74$; d.f. $=1$; $P<0.2$).

Next, comparisons were made between: (a) the 25 index mothers who had recovered psychiatrically, being at low levels (1 or 2) on the PSE Index of Definition at 19-month follow-up; and (b) their individually matched controls (Tables 2 and 3). Not surprisingly, there were fewer significant differences between these groups than between all the index and control pairs. However, as shown in Table 2, index children still showed less affective sharing than control children ($Z=2.24$; $P<0.05$). There was also a trend for index mothers to show less facilitation ($Z=1.91$; $P=0.056$). In addition, index children showed significantly less sociability to strangers ($Z=2.2$; $P<0.05$) (not shown in the Tables).

Severe chronic social difficulties (SCSD) had been experienced by 14 index mothers as against one control mother (Fisher exact $P<0.001$). These difficulties were mainly to do with marriage, housing, or finance. When the 14 SCSD index mothers were compared with the 35 non-SCSD index mothers, certain significant differences were found. As shown in Table 2, the SCSD mothers showed significantly less facilitation, while their children showed significantly less affective sharing. There was also a trend for SCSD mothers to ignore their children more ($Z=1.74$; $P<0.08$). As shown in Table 3, on global measures the SCSD mothers showed significantly less warmth and significantly less rapport. SCSD children were also more likely to be significantly distressed on their mother's departure (χ^2 4.4; d.f. 1; $P<0.05$).

During the first 19 months of the child's life, severe 'negative impact' life events were experienced by 22 index mothers and by 10 control mothers ($\chi^2=6.7$; $P<0.01$). However, no significant differences were found on any variables between 'life-event' and 'non-life-event' subgroups within index or control groups.

The precise timing of depression was noted at each of the post-natal assessments, thus allowing the total length of depression during the post-natal period to be calculated for each mother (in the index group). This ranged from 1 to 19 months (mean 5 months). The length of the mother's depression was then examined in relationship to each of the mother–child outcome measures at 19 months. Duration of depression was found to be inversely related to sociability ($r=0.32$; $P<0.05$) and positively related to the amount of the child's negative responses ($r=0.27$; $P<0.05$) and there was a tendency for it to be inversely related to maternal facilitation ($r=0.2$; $P<0.1$).

The relationship between parity on mother–child interaction was examined separately within index and control groups. All analyses consisted of primiparous mothers being compared with multiparous ones. Parity was found to have no significant effect within the index group. However, some small differences were noted within the control group. Primiparous mothers were found to show less warmth ($Z=1.9$; $P<0.05$) and a tendency to less facilitation ($Z=1.8$; $P<0.07$).

Logistic-regression analysis

As early maternal post-partum depression (0–3 months), maternal concurrent depression (19 months) and severe chronic social and marital difficulties were highly associated with each other, a logistic regression analysis using GLIM (McCullagh & Nelder, 1984) was conducted. The analysis was carried out within the index group in the prediction of mother–child interaction. One-tailed tests of significance were used because of the prior prediction that all three factors were related to mother–child interaction.

We found that low levels of maternal facilitation were independently predicted by chronic social and marital difficulties ($t=2.6$; $P<0.01$), to a lesser degree by early post-partum depression ($t=1.79$; $P<0.05$) and to a negligible degree by maternal concurrent depression. Similarly, low levels of affective sharing were predicted by chronic social and marital difficulties ($t=1.88$; $P<0.05$) and early post-partum depression ($t=1.72$; $P<0.05$). A low level of the child's sociability with a stranger was independently predicted by maternal concurrent depression ($t=2.23$; $P<0.01$) but not by other factors. The child's distress on the mother's departure was predicted by both chronic social and marital difficulties ($t=1.86$; $P<0.05$) and maternal concurrent depression ($t=1.86$; $P<0.05$), but not by early post-partum depression. Lack of maternal warmth was strongly predicted by chronic social and marital difficulties ($t=2.75$; $P<0.01$), but not by maternal depression, whether post-natal or current. No interactions were found between any of the independent factors. These findings indicate that early post-natal depression, concurrent depression and chronic social and marital difficulties made independent contributions to the prediction of the mother–child interaction.

Discussion

This study yielded two main findings about maternal post-natal depression:

(a) that there was a significant association between maternal depression during the post-natal year and reduced quality of mother–child interaction 19 months after the birth of the child

(b) that this association held (though less strongly) not only for mothers who were still depressed at 19 months, but also for those who had recovered from their depression by then.

Furthermore, logistic-regression analysis confirmed that maternal depression in the first three months, maternal depression at 19 months and chronic marital and social difficulties all had independent effects on mother–child interaction.

These findings raise two important questions. Why is maternal post-natal depression associated with a reduced quality of mother–child interaction? Why does this association persist in some mothers who have recovered from their depression?

Three factors may have an important bearing on the first question: (a) the depressive disorder itself; (b) associated marital and social difficulties; and (c) the child's temperament.

The depressive disorder may itself affect the mother–child relationship because the symptoms directly interfere with the mother's capacity to relate to the child (Rutter & Quinton, 1984). Examples of such symptoms are: sadness; self-preoccupation and social withdrawal; loss of interest, loss of energy, and loss of confidence; and irritability. This idea is consistent with the findings in the present study that depressed mothers showed reduced facilitation and rapport, while their children showed reduced affective sharing. It is also consistent with earlier reports that mothers with depressive disorder showed reduced affection and increased withdrawal and hostility towards the child (Weissman & Paykel, 1974), and that depressed mothers were involved in fewer positive interactions with their children (Cox *et al*, 1987).

The extent of the effects of the symptoms may be related to the duration of the disorder as Radke-Yarrow *et al* (1985) found. In the present study, duration of depressive disorder was accurately established and was found to be related to some aspects of mother–child interaction at 19 months in univariate analyses. It was not possible, however, to examine whether duration had an independent effect for two reasons: the mothers with prolonged depression were also those who were still depressed at 19 months; and the numbers of concurrently depressed women were fairly small.

Marital and social difficulties often accompany depressive disorder, as cause or effect or both. A striking finding in the present study was that logistic interactive modelling showed marital and social difficulties to be the most important determinant of unsatisfactory mother–child interaction. This finding is consistent with earlier reports that marital discord and other social factors have important adverse effects on the child (Rutter & Quinton, 1984; Ghodsian *et al*, 1984) as well as on mother–child interaction (Cox *et al*, 1987).

The child's temperament could also play a part in inducing or aggravating the mother's depression, and in reinforcing her lack of confidence and self-esteem. In the present study no attempt was made to assess this factor.

Turning to the second question (the persistence of reduced quality of mother–child interaction despite recovery of the mother's depression), two of the three explanations mentioned above could still operate. Thus marital and social difficulties might persist (and, as indicated above, logistic interactive modelling showed these to be of prime importance). There is some evidence that marital and social symptoms do persist despite recovery in the mother's symptoms (Bothwell & Weissman, 1977). Similarly, the child's temperament could continue to influence mother-child interaction. Whatever the reasons, there is an interesting parallel among older children, namely that there may be no direct temporal relationship between maternal depression and the child's psychiatric disorder. In a study of older children whose parents had a variety of psychiatric disorders, Rutter & Quinton (1984) found no relationship between the "ebb and flow of parental symptoms in the course of disorder in the children".

Despite our finding of an association between maternal depression and reduced quality of mother-child interaction, the considerable variability in the group of depressed mothers should be stressed. Some of the depressed mothers had good interactions with their children, and some were rated highly for warmth. Moreover, reduced warmth was not associated with depression but only with severe and chronic marital and social difficulties.

What are the practical implications of the findings? In this study few of the mothers had seen a psychiatrist or other specialist. Therefore any help is probably best given in primary care, for example, by the general practitioner and particularly by the health visitor who is likely to see the mother and child at the appropriate time.

Suitable help would include encouraging and supporting the mother in her handling of the child, and involving the father and other family members in child care (Rutter, 1991). Attention should be paid to marital and social difficulties. Further research is needed to discover what other approaches may be beneficial.

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Appendix

The procedure for assessing the child's sociability to a stranger is given below. Each response by the child is rated on a 1-5 scale as shown and then an overall impression is rated.

(a) Baby's initial reaction to being offered a toy by stranger on mother's lap (score first 10 seconds following initiation of bid).

(1) Fusses, cries or turns towards mother
(2) Refuses toy by looking away, pushing it away, turning towards another toy, or using words (e.g. 'no')
(3) Looks at toy without reaching for it
(4) Tentatively reaches for or touches toy
(5) Accepts toy from stranger with little hesitation

(b) Baby's reaction to the stranger's initiation of a give-and-take exchange on mother's lap (score first 30 seconds following initiation of bid, or two attempts to get a game started).

(1) Crying, fussing or other indications of distress
(2) Refusal to join in by turning towards mother or another toy, turning away from stranger, just looking at stranger, or using words (e.g. 'no')
(3) Initially reluctant, then participates
(4) Immediately joins in and participates with stranger
(5) Actively participates in game with stranger by smiling, initiating exchanges, anticipating give-and-take, changing the game, acting playfully or teasing the stranger, etc.

(c) Baby's behaviour when given floor freedom.

(1) Touches mother or requests to return to lap
(2) Approaches or turns towards mother
(3) Stays where she/he is or moves away from mother to play with toys
(4) Approaches stranger
(5) Touches stranger or requests to be picked up

(d) Baby's initial reaction to being offered a toy by stranger on floor (score first 10 seconds following initiation of bid).

(1) Fusses, cries or turns towards mother
(2) Refuses toy by looking away, pushing it away, turning towards a different toy, or using words (e.g. 'no')
(3) Looks at toy without reaching for it
(4) Tentatively reaches for or touches toy
(5) Accepts toy from stranger with little hesitation

(e) Baby's reaction to the stranger's initiation of a give-and-take exchange on the floor (score first 30 seconds following initiation of bid, or two attempts to get a game started).

(1) Crying, fussing or other indications of distress
(2) Refusal to join in by turning toward mother or another toy, turning away from stranger, just looking at stranger, or using words (e.g. 'no')
(3) Initially reluctant, then participates
(4) Immediately joins in and participates with stranger
(5) Actively participates in game with stranger by smiling, initiating exchanges, anticipating give-and-take, changing the game, acting playfully or teasing the stranger, etc.

(f) Overall impression of sociability.

(1) Quite unfriendly, fussy, fearful
(2) Between 1 and 3
(3) Generally unfriendly, serious, wary
(4) Between 3 and 5
(5) Neutral, neither friendly nor unfriendly
(6) Between 5 and 7
(7) Friendly, positive reaction
(8) Between 7 and 8
(9) Very friendly, outgoing, smiling

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Lithium: Long-Term Effects on the Kidney

A Prospective Follow-up Study Ten Years after Kidney Biopsy

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Forty-six patients with recurrent affective disorders, who began prophylactic treatment with lithium an average of 20 years previously, were followed up prospectively after a ten-year observation period to assess renal function. Nineteen patients had maintained lithium therapy, and eight patients had died. Tubular function was almost unchanged and patients who had continued lithium had not shown increasing urine volumes, but patients who had received lithium in a single daily dosage at night had a significantly lower urinary output than those on a multiple-dosage schedule. The GFR decreased significantly, but the decline was essentially dependent on increasing age, except in two patients who had developed renal insufficiency Renal function during chronic lithium treatment is related to age, lithium intoxication episodes, pre-existing renal disease, and treatment schedule rather than to duration of prophylactic lithium therapy

The reported histopathological changes in kidney biopsies from a small number of lithium-treated patients with acute lithium intoxication or severe nephrogenic diabetes insipidus (Hestbech *et al*, 1977; Hansen *et al*, 1977) caused widespread concern in the late 1970s and initiated a large number of clinical studies on kidney function and morphology in lithium-treated patients.

There is now general consensus from both cross-sectional (Hullin *et al*, 1979; Vestergaard *et al*, 1979; Coppen *et al*, 1980; Grof *et al*, 1980; Decina *et al*, 1983; Løkkegård *et al*, 1985; Hetmar *et al*, 1986; Gelenberg *et al*, 1987) and longitudinal studies (Vestergaard & Amdisen, 1981; Johnson *et al*, 1984; DePaulo *et al*, 1986; Hetmar *et al*, 1987*b*; Schou & Vestergaard, 1988) that prolonged treatment with lithium may result in a significant urinary concentration defect and polyuria, whereas lithium at non-toxic serum levels has little demonstrable effect on glomerular filtration rate.

However, the retrospective studies gave inconsistent results on the effect of the duration of lithium treatment, and the question of whether maintenance lithium therapy produces long-term kidney damage has yet to be answered definitively. The longitudinal studies, offering a more appropriate design, have generally been limited to two- or five-year observation periods, which may be too short to allow any final conclusion.

In this paper we present the first ten-year prospective follow-up study, using patients as their own controls. Furthermore, as recent studies have suggested that lithium is more harmful to the kidneys when administered in divided daily doses rather than a single daily dose (Perry *et al*, 1981; Plenge *et al*, 1982; Schou *et al*, 1982; Hetmar *et al*, 1986, 1987*a*, 1989), we examined the effect of dosage schedule on renal function.

Method

All 46 patients with a diagnosis of affective disorder (code 296 in ICD-9; World Health Organization, 1978) treated with lithium in the Psychochemistry Out-Patient Clinic in the Department of Psychiatry, Rigshospitalet, Copenhagen, who previously participated in a retrospective functional/morphological kidney study, were invited to take part in a follow-up study ten years later. Twenty-seven patients accepted, and informed consent was obtained from all.

Exactly the same renal functional tests as in the retrospective study (Hetmar *et al*, 1986) were performed during a five-day admission to the Psychochemistry Research Ward which has the facilities to ensure a correct quantitative urine collection. Kidney biopsy was not repeated (Hetmar *et al*, 1987*a*).

Lithium dosage was adjusted by regular serum lithium monitoring. At the time of the initial investigation the optimal range in our clinic was considered to be 0.7–1.2 mmol/l, but gradually the range has become narrower, and at the follow-up study the recommended range was 0.6–0.9 mmol/l for a blood sample taken 12 hours after the last dose. The serum lithium concentrations used for analysis were mean values from the two years previous to the investigation. Lithium levels in serum and urine were determined by atomic absorption spectrophotometry.

Three clearance tests were performed.

(a) Endogenous creatinine clearance: urine was collected quantitatively for three 24-hour periods and creatinine clearance was calculated from the urine and serum creatinine. Median values were used in the statistical analysis.

(b) Plasma ^{51}Cr-EDTA clearance using a single-injection technique (Brøchner-Mortensen *et al*, 1978): glomerular filtration rate (GFR) values were corrected for body surface area.

(c) Lithium clearance (*C*Li): the patients were instructed to empty their bladders at 8.00 a.m. and to collect all subsequent urine for the next 24 hours divided in the following periods: (i) 8.00 a.m.-3.00 p.m., (ii) 3.00 p.m.-10.00 p.m., and (iii) 10.00 p.m.-8.00 a.m. Blood samples were taken at 8 a.m., 3 p.m., and 10 p.m.

*C*Li was calculated as:

$$\frac{C_u \times V}{\Delta C_p \times \text{time}}$$

where C_u = lithium urine concentration and V = urine volume. ΔC_p equals the regressed serum lithium level at the midpoint of the period as expressed by the following equation:

$$\frac{C_p\ (\text{start of period}) - C_p\ (\text{end of period})}{2.3 \times \log\ (C_p\ (\text{start of period})/C_p\ (\text{end of period}))}$$

The mean lithium clearance of the three periods was then calculated considering the length of the period (in hours):

$$C\text{Li} = \frac{7 \times C\text{Li}_1 + 7 \times C\text{Li}_2 + 10 \times C\text{Li}_3}{24}$$

All clearances were expressed as ml/s.

Non-parametric statistical tests (Mann–Whitney, Wilcoxon's test) were used for comparison of direct ungrouped observations. When grouping data, ordinary χ^2 tests for contingency tables have been used or, in case of sparse data, generalised Fisher's exact test.

All statistical tests were two-tailed and significance was set at the 5% level.

Table 1
Characteristics of patients at initial investigation (I) and at a 10-year follow-up (II)

	Examination I	Examination II
Total no. of patients	46	27
on lithium	46	19
off lithium	–	8
Sex		
women	30	19
men	16	8
Mean age (range): years	50.4 (25–74)	58 (36–81)
Mean duration of continued treatment (range): years	8.1 (1–14)	19 5 (11.5–23.5)
Mean serum lithium (range): 12-hour mmol/l, steady-state	0.92 (0.7–1.2)	0.80 (0.5–0.98)
Mean lithium dosage (range): mmol/24 h	29.9 (16–48)	18.9 (12–30)
Lithium preparation: no. of patients		
conventional	44	17
substained release	2	2
Treatment regime: no. of patients		
single dose	26	18
divided dosage	11	1
mixture	9	–

Results

The characteristics of the initial and follow-up sample are shown in Table 1 (examination I and II, respectively). Of the reinvestigated group, 19 patients had maintained lithium treatment, with no breaks exceeding two months. The steady-state serum lithium concentration was 0.80 mmol/l, which was significantly lower ($P < 0.01$) than at the initial investigation (0.92 mmol/l). The reason for the reduction in treatment levels, which has been adopted gradually, was the assumption that lower serum lithium concentrations might be as effective as higher levels while producing fewer side-effects (Coppen & Abou-Saleh, 1988).

Another important treatment variable has been changed within the last four to six years: almost all patients on a divided-dosage schedule gradually changed to a single-dosage schedule in order to reduce renal side-effects in agreement with our previous studies (Plenge *et al*, 1982; Schou *et al*, 1982; Hetmar *et al*, 1986). However, when the influence of dosage schedule on the present renal function was investigated, the original treatment regimes which the patient followed at the time of the initial examination (Table 1) were used.

Of the original group of 46 patients, eight patients had died by 1987. The causes of death were: suicide (3), pulmonary embolus (1), intestinal cancer (1), myocardial infarction (2), and 1 uncertain but with no suspicion of suicide. At the time of death, it was known that four took lithium. None of the patients who had committed suicide were in lithium treatment. These three patients were manic-depressives suffering severe and long-lasting affective episodes, and showing a partial or poor response to lithium. The average age at time of death was 62 years (range 51–77).

Eight patients had stopped lithium treatment after an average of 17.8 years. Side-effects were the main reason for discontinuation: weight gain (1), thirst, complaints of dry mouth and polyuria (2) (one of whom had stopped lithium two months before the follow-up investigation), and 'locked' mood (1). Two patients experienced insufficient therapeutic effect. Two patients discontinued lithium on medical advice; the first patient in relation to neuroleptic malignant syndrome without renal complications, and the second patient developed rapid renal insufficiency after 25 years of continuous lithium treatment (these two patients are discussed in detail under 'Glomerular filtration rate').

No episodes of acute lithium intoxication were recorded.

Five patients refused to take part. Four could not be traced or did not respond to our correspondence. Two patients were admitted to a nursing home in a condition of dementia and were not capable of co-operation, but to our knowledge they had not developed any signs of kidney disease.

Total follow-up participation is 75% if the eight dead and two disabled patients are disregarded.

Table 2
Mean (s.d.) results of initial tests and re-tests 10 years later

	Serum creatinine (mmol/l)		Urine volume (ml/24 h)		Maximum renal concentrating capacity (mosm/kg H_2O)		Endogenous creatinine clearance (ml/s)		GFR (ml/s)	
	initial test	re-test	initial test	re-test	initial test	re-test	initial test	re-test	initial test	re-test
All patients	0.097	0.119***	3056	2939	690	708	1.44	1.20**	1.42	1.25*
	(0.02)	(0.04)	(1346)	(1470)	(174)	(180)	(0.30)	(0.41)	(0.33)	(0.38)
Patients on lithium	0.090	0.11***	3033	2917	607	685	1.44	1.17**	1.38	1.25*
	(0.01)	(0.02)	(1451)	(1615)	(170)	(189)	(0.32)	(0.31)	(0.32)	(0.29)
Patients off lithium	0.094	0.130	3101	2981	704	810	1.44	1.26	1.50	1.25
	(0.02)	(0.07)	(1216)	(1256)	(119)	(123)	(0.2)	(0.5)	(0.36)	(0.56)
Patients on a single lithium dosage	0.093	0.107***	2319	2574	706	711	1.47	1.25*	1.42	1.35
	(0.01)	(0.01)	(548)	(810)	(211)	(202)	(0.32)	(0.37)	(0.33)	(0.30)
Patients on a divided lithium dosage	0.11	0.15*	4375	3803	654	710	1.45	1.19	1.33	1.08
	(0.02)	(0.07)	(1748)	(2170)	(48)	(18)	(0.23)	(0.46)	(0.27)	(0.36)

*$P<0.05$; **$P<0.01$; ***$P<0.001$ (paired t-tests).

Serum creatinine

Table 2 shows that serum creatinine for the total group of lithium-treated patients increased significantly between the two examinations. The average value (0.119 mmol/l) was, however, still within the normal range (0.04–0.13 mmol/l). Table 2 also indicates that serum creatinine increased, although insignificantly in patients who had discontinued their lithium treatment, whereas the increase reached statistical significance in patients who had maintained lithium. Again all average values fell within the normal range.

Both treatment regime groups showed significantly increased serum creatinine values (Table 2), but only those on the divided-dosage schedule had average values above the normal range.

Two patients developed highly increased serum creatinine, in the first case from 0.13 mmol/l to 0.28 mmol/l, in the second from 0.08 mmol/l to 0.20 mmol/l. As these patients showed a general picture of renal insufficiency, these are discussed in detail under 'Glomerular filtration rate'.

Urine volume

Table 2 shows that the average 24-hour urine volume was almost unchanged after ten years of continued lithium administration. Treatment regime had a significant impact on urine volume, but neither was associated with further significant changes in urinary output over the ten years – the initial overall difference had simply been preserved.

Renal concentrating capacity

The maximum renal concentrating capacity assessed by a 26-hour water-deprivation test, which had to be reduced to eight hours in five patients because of weight loss, was essentially unchanged between the two examinations (Table 2). All average values were still below normal ranges except for patients who had stopped lithium. Neither dose regime nor discontinuation of lithium seemed to influence maximum renal concentrating capacity further.

Glomerular filtration rate

Table 2 indicates that the clearance values decreased significantly during the ten-year observation period. This is true for the whole group of patients and most pronounced in patients who had maintained lithium prophylaxis.

In order to examine this result further, all patients were arbitrarily divided into three subgroups with low, median and high GFR values at the initial examination. Nine patients with an average GFR value of 1.05 ml/s (range 0.85–1.20 ml/s) had a follow-up value of 1.04 ml/s; nine patients with an initial value of 1.42 ml/s (range 1.23–1.53 ml/s) had an average value of 1.25 ml/s a decade later, whereas nine patients with high GFR values (1.80 ml/s, range 1.58–2.05) showed a reduction to 1.47 ml/s.

The decrease in GFR values (Δ GFR) between the two examinations was independent of the value at the initial investigation. Apart from two patients with acute renal insufficiency after cessation of lithium, Δ GFR showed a linear progression with time. By using an age-correcting coefficient of 10 ml/min/decade (=0.17 ml/s/decade) (Hetmar & Rafaelsen, 1987), we found a high linear correlation between the predicted GFR and the observed GFR values, independent of continuous lithium administration (Fig. 1).

This was confirmed by the finding of no significant difference in GFR values between patients who continued lithium and patients who stopped lithium treatment (Mann–Whitney, $P=0.74$). As mentioned above, the decreased average values at the follow-up examination in patients who had stopped lithium – as well as in the whole group of patients – were accounted for by marked decreases in

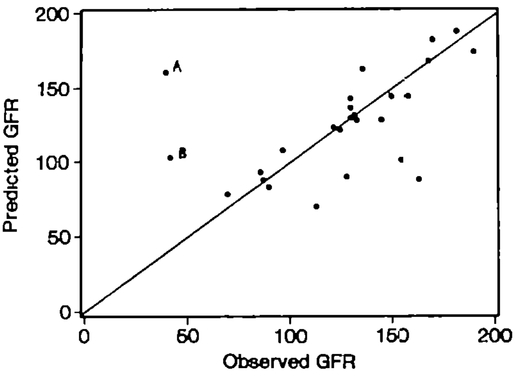

Fig. 1 Glomerular filtration rate in lithium-treated patients: age-corrected prediction versus observed values expressed as ml/s × 10^2. Patients A and B are described in detail in the text.

two patients. Had these two low GFR values (0.42 ml/s and 0.40 ml/s, respectively) been omitted, the mean GFR value would have increased from 1.25 ml/s to 1.32 ml/s.

The first patient was a 64-year-old woman with no past history of renal disease. From 1960 she had had bipolar affective episodes and in 1969 she started prophylactic lithium treatment. Steady-state serum lithium concentration was 0.94 mmol/l; highest recorded value 1.9 mmol/l. In 1978, GFR was 1.76 ml/s and kidney biopsy showed only a slight degree of fibrosis, focally arranged in areas with tubular atrophy. Ten per cent of the glomeruli were totally sclerotic and about 5% of the tubules were atrophied. Lithium was stopped permanently in 1984, when she was admitted with unclear 'neurological' symptoms, but there was no sign of reduced renal function. In 1987 she was readmitted with signs of acute renal insufficiency: serum creatinine was 0.73–0.79 mmol/l and endogenous creatinine clearance was 0.12–0.20 ml/s. Kidney biopsy showed severe acute/subacute tubulo-interstitial nephritis, but no evidence of glomerular or vascular disease. Probably the acute nephrotoxicity was drug-induced, although pyelonephritis could not be excluded with certainty. Treatment with a monoamine oxidase inhibitor was stopped. In three weeks kidney function steadily improved and at the time of discharge from hospital, GFR was 0.7 ml/s and serum creatinine 0.20 mmol/l (Fig. 1, point A).

The second patient was a 72-year-old man with no known renal disease. Since the early 1940s he had had bipolar affective episodes, and from 1962 he continuously received prophylactic lithium treatment. He was classified as a complete responder at a steady-state level of 0.9 mmol/l. About 1975 he developed polyuria (approx. 4 l/24 h) and when entering our study in 1977, GFR was 1.20 ml/s. Kidney biopsy showed that about 5% of the glomeruli were sclerotic and there were areas with atrophic tubules and interstitial fibrosis. From 1984 creatinine values steadily increased. In the same period he developed symptoms of dyspnoea, oedema and angina pectoris, probably due to insufficiency of the mitral valve. No inflammation was found. In 1987 serum creatinine was 0.28 mmol/l and lithium was stopped permanently (Fig. 1, point B).

Renal lithium clearance

Renal lithium clearance decreased from 0.33 ml/s (s.d. 0.095) at the initial test to 0.30 ml/s (s.d. 0.10) at the follow-up examination, but the difference was insignificant. Of the two patients mentioned above, the former did not undergo renal lithium clearance re-test, whereas the latter showed a significantly decreased lithium clearance, from 0.28 ml/s initially to 0.12 ml/s at follow-up.

Discussion

Renal tubular function remained stable in patients continuously treated with lithium for an average of 20 years.

The average urine volume did not change between the two investigations, even though the average serum lithium concentration had been gradually reduced from 0.92 mmol/l to 0.80 mmol/l. Significant associations between serum lithium levels and changes in urine volume (DePaulo *et al*, 1986) and between urine and lithium dosage by body weight (Vestergaard *et al*, 1979) have been reported. However, our results are in agreement with previous findings of no significant correlation between serum lithium concentration and urinary output (Hetmar *et al*, 1986). This may be explained by the treatment regime: urine volume is probably independent of lithium dosage in patients receiving a single daily dose, whereas urine volume increases with increasing dose in patients receiving lithium two or three times a day (Mellerup *et al*, 1985). Interestingly, patients who had taken their lithium in divided daily doses for several years had a significantly higher urine volume (approx. 4 l/24 h) than those on single daily doses, even if the treatment regime had been changed to single daily doses within the last five years (except in one patient).

This persisting difference may reflect tubular atrophy. We have recently shown that the proportion of atrophic tubules was significantly higher in kidney biopsy from the multiple-dose group than in the single-dose group (Hetmar *et al*, 1987*a*). We have reported a significant correlation between 24-hour urine volume and the number of atrophic tubules in patients following a multiple-dose schedule, whereas no relationship was seen in patients on a single-dose schedule (Hetmar *et al*, 1989). This study suggested that renal tubular dysfunction might have a structural basis in a subgroup of lithium-treated patients and may offer an explanation why the average urine volume in this subgroup did not decrease subsequent to change of treatment schedule, as reported in other studies (Perry *et al*, 1981).

Another possibility is that the increased urine volumes are caused by habitual high fluid intake in in response to a dry mouth (associated with tricyclic antidepressants) or on medical advice in order to avoid dehydration during treatment. However, this explanation is not plausible because the increased urine volumes were paralleled by reduced renal concentrating capacities.

Our results add to the increasing amount of evidence (Plenge *et al*, 1981, 1982; Perry *et al*, 1981; Schou *et al*, 1982; Hetmar *et al*, 1986, 1987*a*) that renal structure and function are less affected in organisms receiving lithium in a single daily dose compared with multiple-dose schedule, but the renal influence of different treatment regime has not yet been subjected to prospective studies with random allocation.

No significant changes were observed in maximum renal concentrating capacity during the ten-year observation period, and discontinuation of lithium did not cause any significant increase in renal concentrating capacity. This is contrary to reports of previous studies in which maximum urine osmolality decreased with increasing total lithium exposure (Løkkegaard *et al*, 1985), with increasing duration of lithium (Coppen *et al*, 1980; Vestergaard & Amdisen, 1981; Wallin *et al*, 1982), and with increasing serum lithium concentration (Vestergaard & Amdisen, 1981).

Thus chronic lithium treatment did not produce a progressive tubular dysfunction. Apparently, urine volume as well as renal concentrating capacity reached a steady state, and no further impairment could be demonstrated.

Our study documents a well preserved glomerular filtration rate over 20 years. The decline in glomerular function during the ten-year prospective observation period was essentially dependent on increasing age and could be predicted by using a standard age-correcting coefficient (Fig. 1). This confirms the results of other longitudinal studies (Grof *et al*, 1980; Johnson *et al*, 1984; Amsterdam *et al*, 1985; Vaamonde *et al*, 1986). These studies were however, based on substantially shorter observation periods and mean treatment durations. Løkkegaard *et al* (1985) studied 153 patients treated with lithium for an average of ten years and found a weak but significant correlation between ^{51}Cr-EDTA clearance and treatment duration. They stated that after seven years of treatment practically all GFR values were still within the reference range, and this fact explained why other studies with shorter observation periods failed to show a significant deterioration in GFR. The ^{51}Cr-EDTA clearance regression line reached the normal lower limit after 17 years of treatment (Løkkegaard *et al*, 1985). We were unable to confirm this finding.

After 20 years of lithium treatment only two out of our 46 patients developed renal insufficiency, even though the concentrations of lithium in serum have been relatively high (average 0.92 mmol/l) for several years.

Most cases of histopathological renal changes and impaired function have been found among patients with previous or present episodes of lithium intoxication, which is the most important factor leading to renal damage in lithium-treated patients (Hestbech *et al*, 1977; Hansen *et al*, 1977, 1979).

Other factors may also influence GFR in patients receiving lithium. Our biopsy study showed that the proportion of sclerotic glomeruli and atrophic tubules were significantly related to GFR in patients receiving lithium in divided daily doses, whereas no correlation was found in the group receiving a single daily dose (Hetmar *et al*, 1989). Both patients with renal insufficiency in our study had followed a multiple-dosage schedule. British studies (Cattell *et al*, 1978; Hullin *et al*, 1979; Coppen *et al*, 1980; Page *et al*, 1987) are noteworthy for the absence or very low frequency of renal side-effects observed in patients on long-term lithium treatment. One likely explanation is that many British lithium centres for several years have recommended low serum lithium levels, but possibly it may also be related to the fact that a single daily dose was used in most of these studies.

It cannot be excluded that a subgroup of lithium patients may have latent structural damage without evident renal dysfunction but may eventually develop renal insufficiency after cessation of lithium. Our results emphasise the safety of long-term lithium treatment in the majority of patients but also underlines the importance of treatment schedule and regular monitoring.

In conclusion, progressive impairment of tubular and glomerular function during chronic lithium treatment is related to age, lithium intoxication episodes, pre-existing renal disease, and lithium treatment schedule, rather than to duration of prophylactic lithium treatment.

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Neuropeptide Concentrations in the Cerebrospinal Fluid of Depressed Patients Treated with Electroconvulsive Therapy

Corticotrophin-Releasing Factor, β-endorphin and Somatostatin

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The CSF concentrations of CRF, somatostatin and β-endorphin were determined in nine patients who fulfilled DSM–III criteria for major depression with psychotic features. CSF samples were obtained at baseline in the depressed state, and again after a course of ECT. Concentrations of both CRF and β-endorphin decreased after ECT, while the concentration of somatostatin increased, although the latter difference did not attain statistical significance. The increase in CSF concentrations of CRF and β-endorphin in depressed patients is therefore seen to be state-dependent.

In research on the physiological role of neuropeptide neurotransmitters in the central nervous system (CNS) and their putative role in the pathophysiology of affective disorders and schizophrenia, much interest has centred on corticotrophin-releasing factor (CRF), the 41-amino-acid hypothalamic peptide that regulates the secretion of adrenocorticotrophin (ACTH) and other pro-opiomelanocortin-derived peptides of the anterior pituitary (Nemeroff & Bissette, 1986). When injected directly into the CNS, CRF decreases food consumption and sexual receptivity (Sirinathsinghji *et al*, 1983), and alters locomotor activity (Koob & Thatcher-Britton, 1985). In conjunction with the hypercortisolaemia commonly observed in depressed patients, these findings suggest that CRF is hypersecreted in depressed patients (Gold *et al*, 1984; Nemeroff, 1988). This hypothesis is supported by findings of: elevated CSF concentrations of CRF in drug-free depressed patients when compared with controls (Nemeroff *et al*, 1984; Banki *et al*, 1987; France *et al*, 1988; Arato *et al*, 1988); a blunted ACTH response to intravenously administered CRF (Gold *et al*, 1984; Holsboer *et al*, 1984); and a decreased CRF receptor number in the frontal cortex of suicide victims (Nemeroff *et al*, 1988*a*).

Somatostatin is a tetradecapeptide known to inhibit the secretion of growth hormone and several other anterior pituitary hormones. Somatostatin and its receptors are also distributed throughout the mammalian CNS (Maurer & Reubi, 1985). CSF concentrations of somatostatin are reduced in drug-free depressed patients (Rubinow *et al*, 1983; Bissette *et al*, 1986; Davis *et al*, 1988).

While there is no proof that β-endorphin neurones play a direct role in psychiatric disorders, these cells are known to be responsive to stress and may participate in the expression of various psychiatric symptoms (Akil *et al*, 1986; Nemeroff & Bissette, 1986; Berger & Nemeroff, 1987).

We sought to determine whether clinical improvement in depressed patients treated with electroconvulsive therapy (ECT) is associated with alterations in CSF concentrations of CRF, somatostatin and β-endorphin.

Method

Nine patients (two men, seven women) fulfilling DSM–III criteria for major depression with psychotic features (American Psychiatric Association, 1980) were studied at University Hospital, Stony Brook. The 30-bed in-patient psychiatric unit is part of a large, suburban teaching-hospital complex, which serves as the main tertiary-care centre for a population of two million. The in-patient service is a main teaching unit of a psychiatric department, and the study of new treatments is accepted as a departmental mission: it is not unusual for more than half the in-patients to be part of ongoing research protocols. In this environment we have come to know many affectively ill patients with recurrent illness who view our professionals as their primary psychiatric therapists. It is from this cadre that the patients for the present studies were selected.

The decision to use ECT is made by the treatment team and is based on the severity of the illness, the patient's treatment history, tolerance and response to medications and ECT, and medical status, as well as the wishes of the patient and the family. The ECT unit is well known, and the patients see staff members as both research scientists and clinicians. The ECT consent process follows the guidelines of the American Psychiatric Association (1978). The benefits and risks of the treatment are discussed with the patient and close family members, and they view a professional videotape made at University Hospital. The specific research for which the patient is eligible is described. The written signed consent describes the indications for ECT, its risks and benefits, the experimental procedures (multilead electroencephalography, neuroendocrine tests, CSF measures, neuropsychological tests), and detailed statements of the patient's right to refuse tests or to discontinue treatment or tests at any time. Consent is obtained from both patient and close family members.

For these studies, consent was obtained for two CSF samples, one before treatment, and one when the patient had improved. The consent process; both for ECT and for the various experimental studies, was approved by the Institutional Review Boards of the Health Sciences Center and University Hospital, and by the University Committee on Research in Human subjects.

Once consent was obtained, the pre-treatment tests were done. These included medical, neurological, dental, and anaesthesia assessments; and complete blood count, blood chemistry, electrocardiogram, and multilead electroencephalogram (EEG).

The dexamethasone suppression test (DST) is usually performed about four days after admission in all patients admitted to our unit, and repeated in the four days before the first ECT if there has been an intervening period longer than one week. Cortisol concentrations in plasma were measured from blood samples obtained at 8.00 a.m., 4.00 p.m., and 11.00 p.m. on the day after dexamethasone administration (1 mg at 11.00 p.m.). Plasma cortisol concentrations were measured by radioimmunoassay using commercial kits obtained from Diagnostic Laboratories Inc. Based on our experience with patients at University Hospital, we selected a cut-off point of 5.5 μg/dl as the upper limit for 'normal' values in the post-dexamethasone assays, because this placed 90% of our non-affectively ill patients in the normal DST group (Fink *et al*, 1987).

The Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967) was completed at the time of the DST.

Diagnoses were based on DSM-III criteria and the patients were classified as major depression with psychosis (296.34 or 236.54). All psychoactive medications were tapered on admission or when ECT was being considered. Medications had generally been suspended for four days at the time of first ECT. Benzodiazepines are routinely suspended on admission for affectively ill patients, so that the patients were free of such drugs for at least a week before treatment began. Assessments of mood, behaviour, neuropsychological status, and multilead EEG were done weekly throughout the course of treatment, on a day following a treatment.

The CSF was obtained by lumbar puncture, with patients sitting. Samples were taken in the morning between 7.00 a.m. and 7.30 a.m. with patient activity limited to toiletting. A 22-gauge needle was used and all samples were obtained by MF. Approximately 15 ml of CSF was obtained, and aliquots from the second and third tubes (6–15 ml) were used in the analyses. The first CSF sample was obtained from one to four days before the first ECT treatment. Repeat CSF examinations were done one (six patients), two, three, and four days (one patient each) after an ECT treatment. The time of sampling was determined by the change in mood and behaviour, and the second sample was obtained after ECT sessions 6 and 10 (7 patients), 12 and 14 (one patient each). After the second CSF sampling, four patients had no further ECT; two patients had one, two patients three, and one patient five additional treatments. These were determined by the patient's degree of improvement and our estimate of the need for continuing treatment to maintain it. Thus the mean number of ECT sessions was 8.4 (s.d. 0.95, range 6–14).

Patients received modified ECT with glycopyrrolate (0.2 mg), methohexital (0.5 mg/kg), succinylcholine (0.5 mg/kg), and assisted ventilation with oxygen; electrocardiogram, blood pressure, and blood oxygen saturation were continuously monitored. Seizure duration is monitored by cuff and EEG methods (Gujavarty *et al*, 1987). Seizures are considered adequate when the duration by cuff monitoring was 25 seconds or longer, and the EEG duration at least equal (Fink & Johnson, 1982). Treatments were begun with right unilateral electrode placement, and changed to bilateral placement if the response was not considered adequate after six or seven treatments (Abrams & Fink, 1984). Brief-pulse currents (either MECTA Model D or Thymatron) were used with intensities selected as slightly above threshold. Treatments were given three times weekly.

The CSF concentrations of CRF, somatostatin and β-endorphin were measured by sensitive and specific radioimmunoassay procedures. The concentration of CRF was measured, as previously described in detail (Nemeroff *et al*, 1984; Chappell *et al*, 1986; Banki *et al*, 1987) using an antiserum to ovine CRF generously provided by Dr Wylie Vale (Salk Institute, La Jolla, CA). The method has a sensitivity of 2.5 pg/tube, an IC_{50} (mid-point) of 40 pg, and does not cross-react with other known endogenous peptides. The concentration of somatostatin was measured as described in detail by Bissette *et al* (1986) using an antiserum also provided by Vale; it has a sensitivity of 2.5 pg/tube, an IC_{50} of 100 pg, and shows cross-reactivity only with somatostatin (1–25) and (1–28). Concentrations of β-endorphin were determined by a previously well characterised radioimmunoassay (Cahill *et al*, 1983) which recognises β-endorphin and β-lipotrophin equimolarly. CSF (1.2 ml total) was extracted using Sep Pak cartridges as detailed in Cahill *et al* (1983). The eluate was dried down and resuspended in a radioimmunoassay buffer, allowing the equivalent of 0.6 ml of CSF to be added to each assay tube. Each sample was assayed in duplicate. Artificial CSF was used to verify that the assay was insensitive to salts in the CSF. The midpoint of the assay is 15–20 fmol/tube or the equivalent of 50–70 pg of β-endorphin/tube. It can reliably detect 3–5 pg/tube of β-endorphin or its molar equivalent in β-lipotrophin. It should be noted that there is very little β-lipotrophin-sized immunoreactivity in mammalian brain or in CSF, and the values read represent over 90% β-endorphin-sized material.

Statistically significant differences were sought between CSF peptide concentrations at baseline and after ECT using a paired Student's *t*-test.

Results

The HRSD scores and the post-dexamethasone plasma cortisol concentrations for each of the depressed patients before ECT are shown in Table 1. The mean (s.e.m.) HRSD score for the nine depressed patients was 25.1 (2.6). Of the nine patients studied, eight were DST non-suppressors.

Table 1
Depression ratings and post-DST cortisol concentrations

Patient	Age: years	Sex	Pre-treatment HRSD score	Post-dexamethasone cortisol 8 a.m.	4 p.m.	11 p.m.
1	29	F	39	3.3	1.0	1.0
2	53	M	18	12.5	7.3	–
3	64	F	25	6.6	12.6	2.5
4	28	F	19	19.5	6.9	14.5
5	58	F	19	5.0	7.1	3.3
6	78	F	23	6.0	12.3	6.4
7	69	F	38	8.5	17 1	3.6
8	69	F	19	11.2	18.4	8.7
9	58	M	26	8.0	5.0	13.5

The CSF neuropeptide data, expressed as pg/ml, are presented in Fig. 1. The mean (s.e.m.) CSF concentration of CRF in the depressed patients before ECT was 81.5 (8.8) pg/ml. This concentration is clearly elevated compared with normal controls studied in our previous investigations, who had group mean CSF CRF concentrations of 40–60 pg/ml. After a course of ECT, there was a reduction in mean CSF CRF concentrations ($P<0.025$, Fig. 1). Six of nine patients exhibited a reduction in CSF CRF concentrations after ECT.

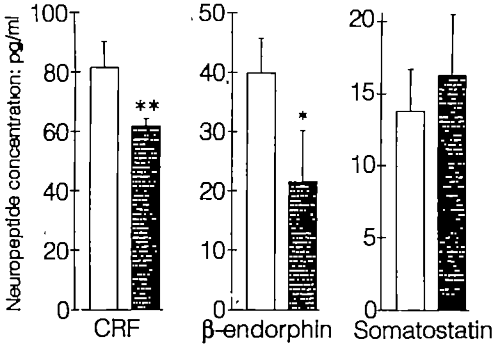

Fig. 1 CSF concentrations (pg/ml) (error bars show s.e.m.) of corticotrophin-releasing factor (CRF), β-endorphin and somatostatin before (□) and after (▨) a course of ECT. There was a significant reduction in the CSF concentrations of CRF ($P<0.025$) and β endorphin ($P<0.05$).

Similar results were obtained with β-endorphin (Fig. 1). Pre-treatment CSF β-endorphin concentrations (mean (s.e.m.)) in the depressed patients was 39.9 (5.8) pg/ml. After ECT, CSF β-endorphin concentrations were reduced to 21.5 (8.7) pg/ml ($P<0.05$). Of the eight patients studied (one sample was lost for β-endorphin measurement), seven patients exhibited a fall in CSF β-endorphin concentrations after ECT.

The CSF somatostatin concentrations before and after ECT exhibited a different pattern to that of CRF and β-endorphin (Fig. 1). After ECT there was a small, statistically insignificant, increase in CSF somatostatin concentrations. Five of eight patients exhibited an increase in CSF somatostatin levels after treatment.

Discussion

Several putative biological markers for affective disorders have recently been identified. These include the DST, the blunted thyrotrophin (TSH) response to intravenously administered thyrotrophin-releasing hormone (TRH), the blunted ACTH response to intravenously administered CRF, reduced platelet ^{3}H-imipramine binding, increased platelet α_2-noradrenergic binding, shortened latency of rapid eye movement (REM), and alterations in CSF concentrations of monoamine metabolites (Asberg *et al*, 1976; Loosen & Prange, 1980; Carroll *et al*, 1981; Gold *et al*, 1984; Holsboer *et al*, 1984; Reynolds & Shipley, 1985; Doyle *et al*, 1985; Langer *et al*, 1986; Nemeroff *et al*, 1988*b*). Whether these biochemical and neuroendocrine alterations represent trait markers or are state-dependent is unclear. An impediment to resolving this issue is the confounding effects of tricyclic antidepressants and monoamine oxidase inhibitors on neurochemical measures, effects that may be unrelated to their therapeutic actions. The study of changes induced by ECT is an ideal paradigm for determining whether one or another putative biological marker is a trait marker, measuring an enduring vulnerability, or conversely is a state marker, reflecting a patient's changing condition. It is ideal because of its documented effectiveness and because there are no confounding pharmacological effects of drugs (Fink, 1979).

It is important to emphasise that what we are studying in the present report is not the acute effects of ECT on CSF neuropeptide concentrations. The first CSF sample was obtained before any ECT. The second was obtained at least 24 hours after the last ECT session. The changes observed are thought to be associated with the changes in behaviour induced by the treatment, and not an immediate post-seizure effect. A parallel is seen in studies of plasma prolactin after seizures. There is an immediate rise in prolactin, with a peak 20–30 minutes after a seizure, and a return to baseline in two to four hours. With successive treatments, the post-seizure rise in prolactin is obtunded, but the baseline level remains the same when measured 24 hours after a seizure.

The alterations in CSF measures were in expected directions. We have previously demonstrated hypersecretion of CRF in depressed patients, as evidenced by increased CSF concentrations. Clinical improvement with ECT is associated with reductions in CRF and β-endorphin concentrations in CSF. We believe that the concentrations of these two peptides represent state markers of depression, particularly psychotic depression.

The somatostatin findings are also of interest. Several groups have reported that depressed patients have low CSF concentrations of this tetradecapeptide (Rubinow *et al*, 1983; Bissette *et al*, 1986; Davis *et al*, 1988). After ECT, there was a small but insignificant increase in somatostatin concentrations in CSF. Of particular interest, however, is the fact that the CSF somatostatin concentrations did not show a pattern similar to CRF and β-endorphin, rendering specificity to the findings observed.

Is the covariation between CRF and β-endorphin in the CSF due to a common regulator which controls both peptides, is it caused by a direct interaction between these two substances, or is it simply the reflection of unrelated consequences of ECT? Basic studies have begun to shed light on this issue. Both neuropeptide systems are intimately associated with responsiveness to stress, not only at an endocrine level, but also within the brain. In addition, results on the relase of β-endorphin from hypothalamic slices show that CRF is a potent releaser of brain β-endorphin (Young *et al*, 1989). Thus, the initial elevation of CRF in depressed subjects may be associated with a concomitant increase in β-endorphin release. Conversely, ECT treatment may lower β-endorphin levels through a number of mechanisms, including the decrease in CRF being liberated. It is likely that numerous other neurotransmitters would participate in this modulation.

Do the changes in CSF peptides represent a non-specific stress response or a differential response of different tissues to the stimulus or the seizure? While many authors have argued that the release of peptides into CSF and blood is non-specific, the reports by Deakin *et al* (1983), Whalley *et al* (1982) and others, cited by Fink (1987), argue for a differential tissue response. The findings in the present study support the view that the release of peptides following induced seizures is a specific tissue response.

Do these observations contribute to our understanding of the therapeutic chain in ECT? There are two prevailing hypotheses of the antidepressant efficacy of ECT (Fink, 1979, 1984). The neurohumoral and receptor hypothesis accepts the changes reported by pharmacologists as the basis for the antidepressant efficacy of antidepressant drugs as the same for the ECT process; that is, the increase in uptake and in receptor activity following induced seizures reversing a reduction in neurohumoral activity seen as the basis for the altered mood (Grahame-Smith *et al*, 1978; Lerer, 1984).

An alternative view argues that altered mood states are associated with a change in brain peptide levels, and that ECT alters these by direct stimulation of brain cells and by altering acetylcholine and norepinephrine brain concentrations which further enhance specific brain tissue responses (Fink, 1979; Fink & Ottosson, 1980). The present observations find differential effects of seizures on three brain peptides, and encourage further study of brain peptides to identify those which may directly affect mood and behaviour.

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The Prognosis of Depression in Old Age

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The 12-month outcome of 103 elderly depressed patients treated by psychiatrists in Perth, Western Australia, was good in 32–47% of cases, depending upon the stringency of the outcome criteria used. A higher than expected mortality was found, especially in men. No clear association was found between 12-month outcome and a variety of clinical variables at index admission, including chronic physical illness, severity of depression, and severe life events during follow-up. As the statistical power of this and other studies is small, it would be unwise at present to draw definite conclusions about the role of possible risk factors in the prognosis of depressed elderly. Vigorous treatment of the depressive illness and adequate after-care are important.

Depressive illness is one of the two most common psychiatric disorders of old age, yet little is known about its prevalence, incidence or natural history (Blazer, 1980). The available longitudinal studies (Post, 1962, 1972; Jolley & Arie, 1976; Murphy, 1983; Baldwin & Jolley, 1986) suggest a relatively poor prognosis. Post (1972) commented that the apparent failure to improve prognosis after the introduction of electroconvulsive therapy (ECT) and tricyclic antidepressants might be an artefact produced by the selection of more intractable cases into hospital in more recent years. Both Post (1978) and Millard (1983) suggested the 'rule of three' for the hospital treatment of depression: no matter what is done, one-third get better, one-third remain the same, and one-third get worse. Murphy (1983) found, in her cohort of 124 patients referred to psychogeriatric services, that the death rate during the 12 months after the index admission was three times that expected for men, and twice that expected for women.

Baldwin & Jolley (1986), using the same criteria for outcome as Murphy (1983), reported that the overall pattern of outcome at one year was dramatically better than that reported by Murphy. Fewer patients had died, many more were well, and the particularly gloomy outcome for those presenting with severe symptoms or delusions, reported by Murphy, was not evident in their cohort. They found considerable agreement between the pattern of outcome identified in their cohort at 42–104 months and that by Post at six years (1962) and three years (1972) after index admission. They concluded that the prognosis for elderly patients who suffered from depressive illness which was severe enough to demand admission was far from gloomy.

The elucidation of the roles of identified risk factors in the prognosis of depression in the elderly is of major clinical importance, and a challenge to psychogeriatric services. Traditional teaching has emphasised the relatively good prognosis of affective illness (Murphy, 1983), and skilled intervention might be expected to produce more dramatic results than is possible with other conditions in the elderly (Baldwin & Jolley, 1986). Murphy (1983) found that severity of initial illness and the patient's continuing physical health were the two most important factors in determining the 12-month outcome of depression in old age. Baldwin & Jolley (1986) disputed the former but confirmed the importance of physical health as a prognostic factor.

This paper describes the 12-month outcome of elderly patients being treated for a depressive illness by psychiatrists in a number of public psychiatric hospitals in Perth, Western Australia. All but five were in-patients. The original cohort was described in detail by Burvill *et al* (1989) and Emmerson *et al* (1989). As pointed out in those papers, the cohort was fairly representative of elderly depressives treated by Perth psychiatrists. No patients refused entry to the study.

The aims of the study were threefold: first, to compare the 12-month outcome of the cohort with the results of Murphy (1983) and Baldwin & Jolley (1986), using the same outcome criteria as in each of those studies; second, to study the effect on outcome of a wide range of factors (both those present at entry to the study, and those occurring during follow-up) using more stringent criteria of outcome than those used in previous studies; and third, to relate outcome to the treatment given during the initial admission.

Method

For entry to the study patients must have been aged 60 years or over, have an adequate command of English, and have a primary diagnosis of DSM-III major depressive illness which had been present for at least four weeks (American Psychiatric Association, 1980). The cohort consisted of consecutive patients meeting these criteria. The details of the research design adopted and the results from the initial assessment of the cohort are given by Burvill *et al* (1989) and Emmerson *et al* (1989). The latter particularly details the methods adopted to measure severe life events and confidants, using the Bedford College Life Events and Difficulties Interview (Brown & Harris, 1978), suitably modified for use in the elderly. Patients were also administered the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960), the Newcastle Scale (Carney *et al*, 1965), and the Mini Mental State Examination (MMSE; Folstein *et al*, 1975). Detailed information relating to the 12-month follow-up (or to time of death) of all 103 patients meeting the entry criteria was also obtained from their medical notes (both in-patient and out-patient), from their general practitioners and in some cases from relatives. Every patient contacted completed the 30-item version of the General Health Questionnaire (GHQ-30; Goldberg, 1972), the Geriatric Depressive Schedule (Yesavage *et al*, 1983), and the Eysenck Personality Inventory (EPI; Eysenck & Edwards, 1964).

Special attention was given to methods of assessing and grading physical illness. Three measures were used: the OARS Physical Health Rating devised by Duke University Centre for the Study of Ageing and Human Development (1978); patients' self-assessment of their health; and a severity measure of physical illness devised by the authors (Burvill *et al*, 1990).

Two classifications of outcome at 12 months were used. First, to allow comparison with the results of Murphy (1983) and Baldwin & Jolley (1986), patients were classified into the following six categories: (a) recovered, (b) relapsed, (c) depressive invalids, (d) continuously ill, (e) demented, (f) dead. The criteria used for all categories except (c) were those described by Murphy (1983). Category (c) (depressive invalids) was added to describe a group of patients who, at follow-up, had definite and moderately disabling symptoms of depression which were not sufficient to reach the criteria for major depression. Following the procedure of Murphy these six categories were collapsed into two broad groups, namely, good outcome (category a) and poor outcome (categories b–f). Thus the good-outcome group comprised those patients continuously well since recovery from the index depression as well as those who had recovered from the index depression, subsequently relapsed and recovered again by the 12-month assessment.

Second, an alternative classification of good and bad outcome was used when examining the relationship between patient characteristics and outcome. The 'good-outcome' category was confined to those patients who had been continuously well since recovery from the index admission: minor symptoms, either continuously or intermittently, were permitted if they had not caused significant subjective distress or functional impairment. The 'poor-outcome' category included all remaining cases. The relationships between individual predictors and 12-month outcome were examined using univariate analyses. In the case of categorical predictor variables (e.g. sex, early v. late onset, severe life event or not) the relationship was indexed by the calculation of an odds ratio, which has an expected value of 1.0 when there is no relationship. In the case of continuous predictor variables (e.g. age, HRSD and Newcastle scores) the relationship was indexed by the familiar correlation coefficient (in this case the point-biserial correlation coefficient). A multivariate statistical analysis and a logistic regression of predictor variables on the dichotomous outcome (good v. poor) were undertaken to take account of relationships between the predictors that would be ignored by the univariate analysis.

Although the 12-month outcome of this cohort of patients is being compared with those reported by Murphy (1983) and Baldwin & Jolley (1986), we wish to emphasise that there were certain differences in the entry criteria into each study. Each required Research Diagnostic Criteria (Feighner *et al*, 1972) for primary depression to be fulfilled. In this and in Murphy's study the initial assessment was made by a psychiatrist or a psychiatric registrar, whereas Baldwin & Jolley chose their cohort retrospectively from an admission register. In this study patients aged 60 years and older were included, similar to that of Post (1962, 1972). Murphy and Baldwin & Jolley used 65 years as the age for entry. We adopted the same policy as that of Post and of Baldwin & Jolley in including depressed patients who had a past or recent history of depression. Murphy chose depressed patients who had been admitted to the area psychogeriatric service for the first time, and although some patients had been depressed in early life, all had been free of depression since the age of 60 years. Both Murphy and Baldwin & Jolley excluded patients with evidence of organic cerebral disorder, whereas we did not.

Results

Patients were aged 60–93 (mean 71.4) years (40% were 60–69 years, 41% 70–79, 19% 80+ years). There were 37 (36%) men and 66 (64%) women; 5% were single, 43% married, 38% widowed, and 14% separated or divorced; 41% lived alone.

At least one severe life event in the 12 months before the onset of depression was reported by 38%, with 24% of these being in the three months before onset. In 54% there was a prior history of depression, with equal numbers with first onset of depression before (early onset) and after (late onset) the age of 60 years. Of the latter, approximately half had had one or more attacks of depression since the age of 60 years. Nine (24%) men (aged 66–81 years) and six (9%) women (aged 66–74 years) were admitted following a serious suicide bid. Diagnostically, using the DSM-III classification, 32% had depressive illness alone, 33% depression with melancholia, and 35% had depression with psychosis. The average score on the HRSD was 23.8. On the Newcastle Scale 17% had neurotic and 83% had endogenous depression. On the MMSE, 77% of patients had a score of 24–30; 22% of 19–23, and 1% of 18; none of these was diagnosed as having a clear-cut dementia at initial assessment.

Six men and five women died one to nine months after initial interview. Only two of these patients had fully recovered from their depressive illness, both of whom died later of heart disease. Two of the remaining nine patients committed suicide, two died from heart disease, one from a stroke, and four from carcinoma. The male mortality rate was 16% and the female rate 8%. Taking into consideration the age profile of the depressed patients, and the differential age-specific mortality rates for Australia for 1987, these mortality rates are almost three times that expected in men and twice that expected in women. However, these mortality rates are based on small numbers and are of questionable statistical significance. The age range of the six men who died was 60–81 years (mean 72.0), and excluding the two who committed suicide was 71–81 years (mean 77.7). The women who died were aged 72–88 years (mean 79.4).

Of the 92 still alive, 68 were seen personally and 11 answered postal questionnaires followed by detailed telephone interviews connected with these. Thirteen patients either refused to be seen or were out of Australia at the time, but these did not differ from the other 90 patients in the following indices on admission: age, sex, severity of depression, number of major life events, number of confidants, or EPI scores. The acute physical illnesses experienced by the whole group in the 12-month follow-up are summarised in Table 1.

The percentage with good outcomes in the Baldwin & Jolley (1986) and Murphy (1983) studies (58% and 35% respectively) were significantly different from each other ($\chi^2 = 13.332$, d.f. = 2, $P<0.05$). Our result of 47% was intermediate, and not significantly different from either of the Baldwin & Jolley or Murphy results. The mortality rate of 11% in our study did not differ significantly from those observed by Baldwin & Jolley (8%) and Murphy (14%). No patients were found to be clear-cut cases of dementia at follow-up. A much smaller percentage of patients (11%) in our study were continuously ill than those of the other two studies (18% and 29% respectively). A similar proportion had relapsed in our study (18%) as in the other two (15% and 19% respectively). By contrast this study had 13% with depressive invalidism, a category not used by either Baldwin & Jolley (for their 12-month follow-up) or Murphy, but used by both Post (1972) and Baldwin & Jolley for their longer-term follow-up studies. This category of patients was clinically much more akin to the 'continuously ill' group than any of the other categories, and adding the invalidism and 'continuously ill' groups in this study gave a figure (24%) between those reported for the continuously ill by Baldwin & Jolley and Murphy (18% and 29% respectively).

Table 1
Acute physical illness during the 12-month follow-up, by outcome

Outcome	Physical illness			Totals
	Life threatening	Other	Nil	
Good	1	7	25	33
Poor	7	13	39	59
Died	9	0	2	11
Total	17	20	66	103

In terms of our second set of outcome criteria, 33 patients (32%) were classified as 'good outcome', that is, had been continuously well, apart from possible minor symptoms, since recovery from the index depression. The remaining 70 patients (68%) were classified as 'poor outcome'. This classification was used to explore relationships between various patient characteristics and outcome.

The results of the univariate and multivariate analyses (Table 2) were almost uniformly negative, except the relationship between having had a severe life event in the three months before the index admission and having a good outcome (OR = 2.76, $P<0.05$). However, in most cases the confidence intervals are wide, and so the failure to reject the null hypothesis of no relationship does not provide a good reason for concluding that there is no such relationship.

The results of the logistic-regression analysis were broadly consistent with the results of the univariate analyses of individual predictors. We were unable to reject the null hypothesis that all of the predictors have a zero relationship with outcome ($\chi^2 = 5.59$, d.f. = 9, $P>0.05$).

Table 2
Relationships between patient characteristics and a good or poor outcome 12 months after index admission

	Measure of association	95% confidence interval
Demographic		
sex	OR = 1.44	0.60–3.60
age	$r=0.06$	−0.13–0.26
Personality		
EPQ-N	$r=-0.07$	−0.12–0.27
EPQ-E	$r=-0.06$	−0.14–0.26
Life events		
three months before admission	OR = 2.76	1.08–7.09*
since index admission	OR = 0.68	0.24–1.92
Social support		
confidant	OR = 1.67	0.55–5.07
living alone	OR = 1.33	0.57–3.07
Psychiatric status		
early onset	OR = 0.94	0.41–2.16
psychotic	OR = 1.30	0.47–3.63
endogenous	OR = 1.67	0.68–4.14
family history	OR = 2.66	0.68–10.06
HRSD score	$r=0.06$	−0.19–0.20
MMSE score	$r=0.03$	−0.16–0.23
Treatment		
tricyclics	OR = 0.84	0.15–4.66
ECT	OR = 1.03	0.44–2.39
Physical illness		
acute (before index admission)	OR = 0.77	0.27–2.18
chronic (before index admission)	OR = 1.18	0.42–3.30
acute (since index admission)	OR = 2.21	0.87–5.59

*$P<0.05$

Discussion

Outcomes and methodologies

Before discussing the results, some clarification of the various outcome measures, and justification for their use, is necessary. We found it necessary to include 'the depressive invalids' category because 13% of our cohort at follow-up had definite symptoms of anxiety and depression, of distressing severity, but not sufficient to fulfil the DSM-III criteria for a diagnosis of major depressive illness. Their symptoms were too numerous and marked to be called 'minor symptoms'. Thus they could not fulfil Murphy's criteria of recovered, relapsed or continuously ill. Many of these patients had these symptoms for many months before the 12-month assessment. The range and severity of symptoms were comparable with those described and categorised by Post (1972) as 'depressive invalidism', and subsequently adopted by Baldwin & Jolley (1986) in describing their three-year follow-up results.

We found the classification into good and poor outcome used by Murphy (1983) to be unsatisfactory, as her category of 'recovered' included those who had relapsed one or more times sufficient to meet DSM-III criteria for depression or 'depressive invalidism', and then recovered sufficiently by the 12-month evaluation to be considered completely well, or to have only minor symptoms. Baldwin & Jolley (1986) expressed major dissatisfaction with the existing outcome classification systems, and used two different classifications to describe their 12-month and three-year outcomes to compare their results for the two periods with Murphy (1983) and Post (1972) respectively. Thus in the major part of this paper, in examining factors associated with 12-month outcome, we have adopted a more stringent criteria for 'recovered', namely, those who have been continuously well since their index admission.

The first aim of this study was to compare the 12-month outcome of the patients with those of Murphy (1983) (35% recovered) and Baldwin & Jolley (1986) (58% recovered): our results were intermediate in both the percentage who had recovered (47%) and who had died. Adding those with 'depressive invalids' and 'continuously ill' gives an outcome group similar to the 'continuously ill' of the other two studies, with a percentage in this group (24%) between the two (18% and 29%). The proportion who had relapsed was very similar in all three studies.

Besides criteria for entry to the respective studies the three cohorts differed in a number of respects. Baldwin & Jolley estimated that their patients were more severely ill and a greater proportion had psychotic symptoms (hallucinations and delusions) than those of Murphy. They were of a similar age distribution. Compared with each of these studies, the patients reported here were less severely depressed, had a smaller proportion with psychotic symptoms, were slightly younger, and the duration of their illness before admission was less. Baldwin & Jolley's cohort had an uncharacteristically high male:female ratio (4:1), compared with that in the other two studies of nearer 1:2. Their study was retrospective from case notes and did not include out-patients, whereas the other two studies were prospective and did include some out-patients.

The reason for the differences (reaching significance comparing the results of Murphy and Baldwin & Jolley) in the proportion in the three studies with a good outcome is not immediately obvious. The usual explanation given for such discrepant results is that the three patient cohorts came from populations which had different prevalences of various prognostic factors, and are therefore not comparable. Murphy, for example, studied consecutive depressed patients referred to a district psychogeriatric service, including both in-patients and out-patients. As such they were a good representative sample of depressed elderly referred to a psychogeriatric service. We considered the 103 patients in this study to be a fair representation of elderly depressed patients admitted to psychiatrists in Perth (Burvill *et al*, 1989) at the time of the study. Only five patients were out-patients, with an outcome comparable to the other patients. By contrast Baldwin & Jolley had so many exclusion criteria that the eventual 100 patients included in their study comprised only about one in five of those described as depressed at initial assessment. Their exclusion criteria seemed very reasonable, but it is possible that with such a rigorous policy, the resulting 100 patients were a group with a potentially better prognosis. The very high proportion of men in their cohort is most unusual and suggests a group of patients not very representative of depressed patients seen by most psychogeriatric services.

It is doubtful that the age or sex differences of the three cohorts would affect outcome comparisons. All studies to date have not shown any sex differences in outcome and any reported differences with age can be accounted for by more physical illness in the older patients (Post, 1986). Baldwin & Jolley (1986) in fact felt that the poor prognosis in men in their study was due to their male patients being more physically ill.

Murphy (1983) reported a poor prognosis linked to severity of the initial depression, and especially to the presence of delusions and hallucinations, but Post (1972, 1986) and Baldwin & Jolley (1986) and this study found no such association. Copeland (1988) reported similar outcome results to Murphy in his three-year longitudinal study of community depressives even though his cohort contained fewer subjects with severe illness. The present study measured severity of depression in several different ways, all of which failed to show a clear association between severity of index depression and outcome. All three DSM–III categories of depression (depression only, depression with melancholia, depression with psychotic symptoms) had a very similar outcome.

Post (1986) concluded, from his survey of the available outcome studies, that duration of illness was the only unequivocal indication of inferior outcome: unremitting depression for more than two years made a good response to treatment highly unlikely, and there was a significant trend for patients who had been ill for less than one year to do better than those who had been depressed for one to two years. In Post's (1962) cohort, 45% of patients were ill for more than one year, in Murphy's (1983) 19%, and in Baldwin & Jolley's (1986) 18%. In our study no patients had been continuously depressed for more than one year before admission. These reported reductions in the duration of illness before admission over the past 30 years probably reflect current increasingly earlier detection and treatment of depression by general practitioners, the earlier referral of more severe and difficult patients to psychiatrists, and the more widespread use of tricyclic antidepressants and other treatments. Baldwin & Jolley failed to find that duration of illness predicted a poor outcome, whereas duration of illness more than 12 months before interview was one of the four factors that Murphy (1983) found discriminated between good and poor outcome.

Mortality

The 12-month death rate in this study was within the range of those reported by of Murphy (1983) and Baldwin & Jolley (1986), with a rate about three times the expected rate in men and twice the rate in women. Murphy (1983; Murphy *et al*, 1988) found a higher 12-month and four-year mortality rate in her cohort of depressed elderly, particularly in men, which was not due to differences in physical health alone. Increased mortality of depressed elderly in-patients has been reported by many authors in the past (Stendstedt, 1959; Kay, 1962; Post, 1972; Jacoby, 1981). Similarly, Kay & Bergmann (1966) and Copeland (1988) have found increased mortality in men in follow-up studies of depressed elderly community subjects. Increased mortality of patients with depression has also been reported in younger groups (Post, 1986; Lonnqvist & Koshenvuo, 1988).

As in this cohort, the various authors have reported that suicide makes only a small contribution to the increased mortality in depressed elderly (Post, 1986). One of the suicides seemed almost inevitable. The patient was admitted to a mental hospital before the index admission, denied all symptoms of depression, was discharged and entered this study after a very determined suicide bid. He continued to deny depression even after an apparent full recovery after ECT, was discharged and committed suicide within a few weeks. The second did not have a previous history of suicide, was well on discharge, but subsequently relapsed and committed suicide after discharge.

Apart from the two suicides, all the remaining nine patients died from acute illness, in keeping with Baldwin's (1980) Oxford record linkage study which showed that depressives have an increased risk of ischaemic heart disease, cerebrovascular diseases, and hypertension. Brown & Paraskevas (1982) reviewed the literature and suggested that depression may present as an early symptom of cancer, antedating the discovery of the malignancy.

Physical health

Murphy (1983) found that the patients' continuing physical health was one of the two most important factors in determining outcome (initial severity of depression being the other). Other authors have reported a positive association between outcome and chronic health problems (Roth & Kay, 1956; Kay & Bergmann, 1966; Post, 1972; Baldwin & Jolley, 1986), although Post (1962) did not find such an association. We found the same amount of chronic physical illness in both the good-outcome and poor-outcome groups. It is not clear why our findings should differ so markedly from those of other investigators, especially as the method of measurement of physical illness was given special emphasis in this study. One possibility is that this cohort was clearly from less disadvantaged environmental surroundings than those in many of the British studies, and had fewer life difficulties of all sorts, including chronic health problems, thus diminishing the potential role of the latter as prognostic indicators. The absence of the contribution of extremes of cold weather in winter months in those with chronic physical illness may have been a factor. That a significant association was found using the patients' own assessments of their physical health is in keeping with reports in the literature (Singer *et al*, 1976; La Rue *et al*, 1979; Mossey & Shapiro, 1982).

By contrast there was an excess of life-threatening, *acute* physical illnesses in the poor-outcome group. Of the 11 who died, 9 had had life-threatening illnesses after admission, and of the 59 still alive at follow-up and with a poor outcome, seven had a life-threatening illness, while only one of those with a good outcome had a life-threatening illness.

Treatment

The finding that no particular treatment, or combinations of physical treatment, was associated with outcome should not be interpreted as meaning that one form of treatment, such as tricyclic antidepressant, is just as effective as another treatment, such as ECT. Rather the finding is more likely to indicate that appropriate physical treatment was given depending upon the clinical status of each patient. Confirmation of this interpretation is found in the fact that the more severely depressed patients were more likely to be given ECT, and that those given ECT were likely to be in hospital longer than the other patients.

Baldwin & Jolley (1986), in attempting to explain the better 12-month outcome in their cohort of patients than that of Murphy (1983), suggested that the patients in their study were more vigorously treated. The most clear-cut difference in treatment that they were able to identify was in the proportion of patients given ECT. Of Baldwin and Jolley's patients, 48% were given ECT, compared with 16% in Murphy's cohort (38% in this study). The justification of more ECT cannot be based on the difference in severity of depression in the three cohorts, as that of Baldwin & Jolley were, on the average, more severely depressed than in the other two, and the patients in this study were the least severely depressed. Is it possible that the very poor outcome of Murphy's patients with depressive delusions was because ECT was used much less often than in the other two studies? This might explain the positive relation between severity of depression and poor outcome in Murphy's study, which was not found in the other two. Only 5% of the 20 patients in Murphy's study who were treated with ECT had a good outcome. This relatively poor outcome following ECT may be due to the treatment being given to only a restricted number of patients, many of whom had poor outcome prospects, rather than to a failure of ECT to be helpful in treatment. Alternatively the number of ECT sessions given to each patient could have been smaller than in the other two centres. It is interesting that the best outcome in this study was in those few patients who were treated with a combination of tricyclic antidepressants, ECT and lithium carbonate (a fairly vigorous treatment regime).

Godber *et al* (1987) reported 163 depressed patients treated with ECT (representing 20% of all depressives admitted) whom they followed up for three years. They tended to use ECT early in the illness when delusions were present. Those making initial full recoveries fared best subsequently. They concluded that ECT has an important and generally underestimated place in the management of depression in the elderly. Rifkin (1988) reviewed nine studies comparing the treatment of depression by ECT and antidepressant drugs in all age groups, finding most were methodologically deficient and no conclusion was possible as to which treatment was the more effective. Benbow (1989), in a review of the use of ECT in the treatment of depressive illness in old age, concluded that old age may be associated with a better prognosis with ECT than that in younger age groups. She stressed that, in view of the continued chronic morbidity of depressive illnesses despite modern antidepressant drugs, it is important to review the use of such treatment in the aged in order to minimise the possibility of patients being denied effective treatment because of preoccupation with temporary side-effects, exaggerated potential hazards, or media pressure.

Clinical experience would suggest that follow-up treatment after discharge from hospital, or recovery from the initial illness, is also very important in long-term outcome. Godber *et al* (1987) reported that the long-term morbidity of a relapsing illness can be minimised by enthusiastic follow-up with early detection and treatment of relapses. Baldwin (1988) has argued strongly for the permanent after-care of elderly depressives, based on their high known relapse rate, that these relapses are often undetected without planned after-care, and that further treatment is often successful.

None of the three outcome studies considered in this paper addressed the question of after-care treatment. The prophylaxis and after-care of elderly depressives has yet to be systematically evaluated (Baldwin, 1988).

Social circumstances

Similar to the finding of Murphy (1983), no significant difference in outcome was found between those living alone and those not. However, a more sensitive measure of social support is the patient's subjective view of whether or not he/she has an intimate confiding relationship. This is correlated with marital status but does not coincide with it (Murphy, 1983). Again the results in this study were similar to those of Murphy, with a higher proportion of those who originally reported a good confidant in the good-outcome group, but the differences were not statistically significant in either study. Her results led Murphy to conclude that there was no evidence that an intimate relationship protected against relapse in the face of continuing life stress.

Noteworthy was that of Murphy's 77 patients on whom relevant information was available at follow-up, 13 changed their rating regarding the presence or absence of a good confidant between the index admission and follow-up. In all but two of these cases the change was from a rating of poor/no confidant to that of good confidant, a change which occurred mostly in those patients who were relatively well at follow-up. This suggests that some depressed patients are likely to view their immediate social environment rather more negatively than when not depressed. A similar change in the responses to the EPI of those patients who were no longer depressed was also noted at follow-up. This finding raises some serious doubts about the reliability of self-ratings of the presence or absence of confidants in depressed patients, and of the theories based on such measurements.

Statistical power

Low statistical power is a problem in each of the outcome studies of depression in the elderly considered in this article (Post, 1972; Murphy, 1983; Baldwin & Jolley, 1986; Burvill *et al*, 1989), especially when examining relationships between the predictor and outcome variables. When the outcome is a dichotomous one, such as good or poor outcome, and many of the predictors are also categorical (e.g. sex, diagnosis, severe life event or not), the probability of detecting all but the strongest correlation between predictors and outcomes is small. Such statistical power is largest when the sample is evenly divided between categories or the predictor variables, and even then it is poor. It declines steeply as the proportion of cases in the least frequent categories declines. For example, Cohen's (1977) power tables indicate that a sample size of 100 provides only 17 chances in 100 of detecting a 'small' relationship between predictors and outcome, and such a size of relationship is what one would expect between the individual predictors and a dichotomous outcome.

The major consequence of the low statistical power is that the studies that have been conducted to date will have a small chance of detecting a relationship between such predictors and outcome. This is the most probable explanation of our failure to find any such relationships. To overcome this problem much larger cohorts would be needed. Cohen's tables suggest that a sample size of 200 (or more) would be necessary to provide an 80% chance of detecting a small to medium sized relationship between individual predictors and a dichotomous outcome.

Conclusion

The 12-month outcome of depressive illness in the elderly is not as gloomy as that reported by Murphy (1983) and expressed by Millard (1983) in the 'rule of three'. The criteria of outcome used by various authors is unsatisfactory, and the use of different criteria can result in quite different results. The results underline the fact that a high proportion of depression in the elderly, as in the young (Kiloh *et al*, 1988; Lee & Murray, 1988), either proceeds to various degrees of chronicity, or is recurrent, and carries a higher than expected mortality rate.

The results give some support to the contention of Baldwin & Jolley (1986) that the vigour and adequacy of treatment of the depressive illness in the index admission has an important influence on outcome. Review of existing knowledge would indicate that early detection and the vigorous treatment of presenting depressive illness, including use of ECT, together with close attention to after-care, offer the best prospects for improving the prognosis of depressive illness.

The study failed to find any significant relationship between outcome and nearly all the predictor variables examined, although the small sample size may have influenced this finding. We should be wary about drawing any final conclusions about such relationships until these have been examined in prospective studies of much larger patient cohorts than those conducted to date. Severity of depression, including psychotic symptoms, was not found to be a predictor of outcome, as reported by Murphy (1983), and no clear relationship was found between outcome and chronic physical illness, contrary to the findings of many investigators. Two significant associations were a good outcome in those who had a severe life event in the three months immediately before the onset of the index depressive illness, and a clear association between severe acute physical illness at index admission and death during the first 12 months.

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Depression and Previous Alcoholism in the Elderly

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A prospective study of male in-patients over 55 years old who met Feighner criteria for non-bipolar depression was performed to determine if a previous history of alcoholism significantly influenced treatment or response to treatment. Among 58 subjects with complete follow-up information, the 16 who had a history of alcoholism had a presentation at index which differed from that of the non-alcoholics, and on follow-up they clearly had more chronic illness. This elderly sample with alcoholism resembles 'neurotic-reactive' depressives described in younger samples, and supports a past history of alcoholism as being a risk factor for chronicity of depression on follow-up in the elderly population.

Alcoholism and depression have long been noted to be seen in the same individuals (Amark, 1951). Typical research concerning the co-occurrence of these disorders has involved looking at the frequency of depressive symptoms (Pottenger *et al*, 1978), as well as acute pharmacological response to antidepressants (Ciraulo & Jaffe, 1981), in alcoholics presenting for detoxification. Further, the influence of a family history of alcoholism upon course and outcome of depression in individuals without alcoholism has been studied (Winokur, 1978). While these factors are all important, the question of what influence a past history of alcoholism has upon the clinical presentation, treatment, acute response and outcome in patients presenting with depression has been neglected in the literature. Giles *et al* (1987) did consider this issue as part of a larger study of subtypes of secondary depression, and found that depressions beginning after alcoholism in abstinent alcoholics were clinically very similar in their presenting symptoms, Hamilton Rating Scale for Depression scores, latency of rapid eye movement, and rates of dexamethasone non-suppression. Indeed, the only real difference noted in that study was that the primary depressives had a younger onset of depression, but it did not consider treatment outcome or follow-up course of illness for the two groups.

Rates of depression among alcoholics have been reported to vary between 3% and 98% (Keeler *et al*, 1979), with the results of the majority of studies ranging between 30% and 60% (Bowen *et al*, 1984). Such studies however have considered subjects who were still actively alcoholic, and the rates are heavily dependent upon whether specific criteria versus rating scales are used for the diagnosis of depression (Petty & Nasrallah, 1981). Treatment studies of depressed alcoholics have generally indicated a poor response to tricyclic antidepressants (Viamontes, 1972; Shaw *et al*, 1975), but such studies use samples with mixed alcohol and depressive symptoms, presenting primarily for alcoholism treatment.

The present study differs from previous studies in that it looks at an elderly population of depressed in-patients seeking treatment for depression, but in whom a past history of alcoholism was recorded. The purpose of this paper is to evaluate whether such a past history of alcoholism in elderly depressives who have not displayed recent problem drinking is clinically relevant.

Method

This project consisted of an historical prospective study of male non-bipolar depressives over 55 years of age, admitted to hospital at the Iowa City VA Medical Center between 1978 and 1980. Subjects were included if they met Feighner criteria (Feighner *et al*, 1972) for a major depressive episode, regardless of alcohol history beyond two years before admission. Feighner criteria for alcoholism was next assessed in all such depressives. Of 62 subjects who met criteria for major depression, the 58 who were available for follow-up interview three to five years later are considered in this report.

Data regarding the index depressive episodes were all obtained from the clinical records through careful chart review. The follow-up interviews were conducted primarily by telephone, using a semistructured format to obtain the clinical history since index discharge. At the time of this follow-up interview, review of the index history was conducted along with a survey of family history for psychiatric illness. All ratings were completed by a research assistant blind to the purpose of the study.

Categorical data were analysed using χ^2 statistics, or a Fisher's exact test when expected cell counts were <5. Continuous data were analysed using Student's *t*-tests. Variance testing was conducted using an *F* test, and where significant differences were noted Cochran's approximation was used for significance testing.

Results

All subjects were elderly Caucasian men and 16 (27.5%) had a past history of alcoholism. In four of the alcoholic

Table 1
Demographic data

	Alcoholism history ($n=16$)	No alcoholism history ($n=42$)	P
Mean age at index admission: years (s.d.)	57.7 (4.8)	62.6 (8.6)	0.01[1]
Age range at index admission: years	55–67	55–84	
Mean age at onset of depression: years (s.d.)	40.7 (14)	50.6 (13)	0.01[1]
Mean age at first admission for psychiatric reasons: years (s.d.)	44.1 (14)	54.6 (12)	0.009[1]
Marital status on admission: % separated or divorced	31.2%	7.1%	0.03[2]
Index chart diagnosis of reactive depression	25%	8%	0.083

1. t-test, d f. = 56
2. Fisher's exact P.

Table 2
Index admission treatment (including electroconvulsive therapy (ECT) and tricyclic antidepressants (TCAs)) and acute response

	Alcoholism history ($n=16$)	No alcoholism history ($n=42$)	P[1]
Index treatment			
ECT	13%	43%	0.027
TCA alone	81%	40%	0.006
non-ECT/non-TCA	6%	17%	NS
Acute treatment response:			
full	25%	41%	
partial	62%	57%	NS
none	13%	2%	
Discharge medication·			
TCA alone	88%	57%	0.027[2]
MAOI/TCA + Li^+/ neuroleptic-TCA	0%	14%	
none	12%	29%	
Mean duration of index admission: days (s.d)	21.9 (12)	49 (53)	0.004[3]

1 Fisher's exact P, except as otherwise noted
2 Comparison is TCA alone v. any other treatment
3 t-test, d f = 56

subjects the depression was temporally first, and in the remaining 12 the depression came second. All subjects were admitted at index with the diagnosis of major depression, and had not actively displayed problem drinking for at least two years. Rates of total abstinence from alcohol in both the alcoholic and non-alcoholic groups could not be reliably ascertained at index by the study methods employed.

Table 1 summarises the demographic features of the two groups. The subjects with a history of alcoholism were younger at index admission and at first admission to hospital, and had a younger age of onset of depression. They were more likely disrupted in their marital status (divorced or separated), and tended to be more frequently given discharge diagnoses of reactive-type depressions.

At index admission, the group with past alcoholism had had more frequent past admissions to hospital. Of those with a history of alcoholism, 37.5% had less than three previous psychiatric admissions compared with 71% of those with no alcoholism (Fisher's exact $P=0.02$). Both groups presented with many of the typical depressive symptoms enumerated in the Feighner criteria (>6 such symptoms in each group), but the group with no history of alcoholism had more delusions (31% v. 6%; Fisher's exact $P=0.045$), and more agitation or retardation of motor activity (95% v. 75%; Fisher's exact $P=0.043$), while the group with a past alcoholism diagnosis reported more suicidal ideation (88% v. 59%; Fisher's exact $P=0.038$). Duration of depressive symptoms and the percentage with concurrent medical illness upon index admission did not differ between the groups.

Treatment at index admission in the two groups was clearly different (Table 2). A higher proportion of the group with no alcoholism history received electroconvulsive therapy (ECT) as well as treatment with agents other than tricyclic antidepressants (TCAs). The group with past alcoholism more often was treated with TCAs alone and was admitted to hospital less than half as long as the non-alcoholic group, but clinical ratings of full v. partial v. treatment non-response were equivalent.

Table 3 summarises clinical follow-up data obtained about four years after the index admission. While both groups had an equal duration of follow-up, the alcoholic group had more frequent readmissions to hospital, and took antidepressant medications for longer. The frequency of out-patient visits was similar in both groups. Among the 16 subjects with a history of alcoholism, eight had returned to some alcohol use, and two of these had gone on to have additional alcohol-related problems. Despite the group with an alcohol history being younger at the time of index evaluation, 25% were dead by follow-up compared with only 12% of the non-alcoholic group. This was not statistically significant because of the small numbers involved, but clinically appears as a striking difference. The degree of recovery outlined in Table 3 can be summarised by saying that the alcoholic group felt as though they had never reached as satisfactory a recovery as the non-alcoholic group. The alcoholic group continued to be more disrupted in marital status, and more frequently lived alone.

The group with an alcoholic history (despite being younger) were more dysfunctional occupationally on follow-up, being less likely to return to work (37% v. 69%; Fisher's exact $P=0.02$) and having more medical disabilities (46% v. 10%; Fisher's exact $P=0.004$). The percentage of each group disabled by psychiatric illness was not significantly different (13% v. 21%).

Table 3
Follow-up course and recovery

	Alcoholism history ($n=16$)	No alcoholism history ($n=42$)	P[1]
Course			
Duration of follow-up: months (s.d.)	49 (15)	50 (18)	NS
% admitted 3+ times	25%	0%	0.004
No. months in hospital: mean (s.d.)	1.1 (2)	1.6 (4)	NS
% taking antidepressants at follow-up	37%	42%	NS
% ever tried to stop antidepressants by follow-up	68%	69%	NS
% having taken antidepressants <50% of the time since index	25%	78%	0.001
% with >3 outpatient visits yearly since index	60%	51%	NS
% fully alcohol abstinent at follow-up	50%	83%	0.013
% deceased	25%	12%	NS
% deaths natural	50%	80%	NS
Recovery			
Maximum recovery within 1 month of index discharge	25%	55%	0.05
Maximum ever recovery of one symptom or less	25%	78%	0.001
% ever reporting social recovery	62%	83%	0.09
Overall improvement judged on follow-up to be partial or none from index	43%	12%	0.014
Follow-up marital status: divorced or separated	37.5%	4%	0.003
Living alone at follow-up	46%	15%	0.02
% living alone for >50% of follow-up period	40%	12.5%	0.03

1. Fisher's exact P.

Of the depressives with a personal history of alcoholism 7/16 had a first-degree family member affected with alcoholism compared with only 7/42 of the subjects without a personal history of alcoholism (Fisher's exact $P=0.038$). When all families without alcoholism are evaluated for affective disorder, 0/9 in the alcoholic group v. 8/35 in the non-alcoholic group had a first-degree family member with a history of affective disorder (Fisher's exact $P=0.02$).

Discussion

This report should be considered preliminary in nature, and further studies with larger samples are needed to support the concept of subtyping elderly depressives in terms of past history of alcoholism. Such studies should also consider using multivariate analysis techniques to help describe the relationship between the multiple variables which may be of importance in such individuals. With these limitations in mind, this study does suggest that depressed male elderly patients with a past history of alcoholism differ from depressives without such a history. Such subjects appear to present with a depression which is less 'endogenous', that is less agitated/retarded and less psychotic, for which more reactive-type diagnoses are given. Treatment may be less aggressive as judged by reduced rates of utilising ECT or non-TCA treatment modalities, but acute response as rated by the clinical record appears equal. Despite these optimistic features, the pattern of their illness tends to be more chronic. Features of 'neurotic-reactive' depression as outlined by Winokur (1985) appear to persist in this group of elderly depressives with a history of alcoholism despite the small rate of return to problem drinking during the follow-up period (2/16). Such features include frequent readmission to hospital, more chronic continuation of antidepressant medications, more suicidal ideation at index, and less clear remission of depressive symptoms at follow-up.

Several explanations of this pattern are possible. The depression in those with previous alcoholism is clearly less familial, and thus may be aetiologically distinct (i.e. a phenotype). Another explanation might be that the alcohol exposure induces organic changes of the central nervous system which persist, and predict poor response to treatment. Certainly, personality features not measured in this study could have large influences. Another consideration may be that the group with alcoholism in this study was inadequately or undertreated at the time of the index presentation. While this is clearly possible, global ratings from blind review of the records would not indicate that clinically perceptible differences in responses were present by index discharge.

Although at present it cannot be said from this study why these groups differ, it can be observed that elderly depressives with past alcoholism represent a clinically distinct group of depressives that are not uncommonly encountered. It further appears that present treatment of such depressives is less successful than in elderly depressives without a history of alcoholism. Clinical studies of whether treatments other than TCAs alone, improved social support or other measures would be more effective may also be warranted.

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Brain Lesions and Cognitive Function in Late-Life Psychosis

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Twenty-four patients who developed their first psychotic episode after the age of 45 were studied with MRI and comprehensive neuropsychological testing and compared with 72 healthy elderly subjects. The patients demonstrated more clinical abnormalities on MRI, were more likely to have large white-matter lesions or metabolic illness, and did more poorly on many neuropsychological tests, particularly those testing frontal-lobe and memory abilities. We conclude that structural brain injury is commonly associated with the late onset of psychosis. Neuroimaging investigations are a valuable component in the evaluation of this patient group.

There has been a century-long controversy regarding the diagnosis and aetiology for patients who develop psychosis late in life. Kraepelin (1919) observed that approximately 10% of patients with a schizophrenic-like illness developed their symptoms after the age of 40, and he coined the term 'paraphrenia' to describe these patients. Although the concept of paraphrenia was not universally accepted, Roth (1955), Kay & Roth (1961), and Post (1966) helped renew interest in the phenomenology and aetiology of late-life psychosis (LLP). Roth (1955) differentiated these patients with LLP from those with 'senile', 'arteriosclerotic', and 'confusional' psychosis, and noted that the paraphrenics had better long-term prognosis, including a lower mortality rate.

More recent studies have suggested that some patients with LLP have structural or metabolic brain disease as a contributing, if not causal, factor in their illness (Leuchter & Spar, 1985; Miller *et al*, 1986; Holden, 1987). For example, Miller *et al* (1986) described four non-demented, non-neurologically impaired patients with LLP in whom subtle evidence of structural brain injury was evident with computerised tomography (CT). The authors noted that in previous decades the structural lesions might not have been detected during life.

To explore further the relationship between structural brain injury and LLP, we did psychiatric, neurological, and neuroimaging examinations comparing patients with LLP with normal elderly controls. Additionally we administered a comprehensive neuropsychological (NP) battery, because neither the NP characteristics of patients with LLP nor the relationship between NP function and abnormalities on neuroradiological tests in this population had been investigated systematically (Cullum *et al*, 1988).

Method

Prospective patients were recruited from the in-patient wards and out-patient clinics of a university-affiliated general hospital. In addition, patients were referred to us by clinicians who knew we were studying patients with LLP. Patients with the onset of psychosis after the age of 45 were evaluated. The history was obtained from the patient and, when possible, was corroborated by interview of family and review of medical records. When there was doubt regarding the age of onset, the patient was not studied. Patients were evaluated both clinically and with the Structured Clinical Interview for DSM–III–R (SCID; Spitzer & Williams, 1986).

Patients with the DSM–III–R (American Psychiatric Association, 1987) diagnosis of schizophrenic disorder (late-onset type), schizophreniform disorder (of at least one-month duration), delusional disorder, and psychosis, not otherwise specified (NOS) (of at least one-month duration), were included for study. Most had been on neuroleptics for several days to weeks before evaluation.

The patients had physical, neurological, and laboratory examinations, and a magnetic resonance imaging (MRI) scan. CT was done in three patients in whom MRI could not be done. The Brief Psychiatric Rating Scale (BPRS; Overall & Gorman, 1962) was completed and the Mini-Mental State Exam (MMSE; Folstein *et al*, 1975) was used to quantify cognitive impairment. Patients were excluded if the MMSE score was less than 24. Other exclusion criteria were a history of drug or alcohol abuse, stroke, epilepsy, Parkinson's disease, or evidence of hemimotor or hemisensory deficits. All patients were fluent in English.

The patients were compared with controls recruited through an advertisement in a local paper announcing that elderly people were wanted for a study of normal ageing. Controls were studied with the same protocol used for the patients. They were free of current or past psychotic, major affective, alcohol or substance abuse disorders, scored over 24 on the MMSE, and passed the same exclusion criteria as did the patients. Subjects gave written informed consent before participation in the study.

The battery of NP tests included the Wechsler Adult Intelligence Scale – revised (WAIS–R) (Satz–Mogel format), the logical memory (LM) and visual reproduction (VR) subtests of the Wechsler Memory Scale (WMS), the Rey–Osterreith complex figure test (RO), the Wisconsin Card-Sorting Test (WCST), the Stroop test, auditory consonant trigrams (ACT), verbal fluency (FAS), and the Warrington Recognition Memory Test (RMT–W (words), RMT–F (faces)). These tests provided information about general intelligence, attention (WAIS–R digit span), memory (LM, VR, RMT–W, RMT–F), frontal-lobe function (WCST, Stroop, ACT, FAS), and visual-spatial skills (RO). Test variables used for analysis have been described in previous work (Lesser *et al*, 1991).

A Picker scanner with a 1.5 tesla superconducting magnet was used for the MRI scans. Axial, coronal and sagittal scans were performed – the protocol details are described previously by Lesser *et al* (1990). Axial scans were done parallel to the canthomeatal line from the skull base to the vertex in 10 mm sections. A repetition time (TR) of 2000 ms was employed, and echo times (TE) of 20 ms and 100 ms were used to give scans that were T_1- and T_2-weighted respectively. The scans were read for clinical diagnoses by two independent raters (a neuroradiologist and a neurologist) blind to subject status.

Scans were transferred to magnetic tape, and specific measurements were made at a viewing console where a cursor under control of a joystick was used to outline regions of interest. The areas of the cerebral hemispheres and lateral ventricles were measured by a computer program which calculates area to 0.01 cm^2. These were measured on the same T_1-weighted axial section through a standardised slice showing the largest extent of the bodies of the lateral ventricles. A ventricular:brain ratio (VBR) was calculated as described previously (Lesser *et al*, 1990). Two independent observers each read MRI scans from 15 randomly selected cases. Intraclass correlation for their VBR measurements was excellent ($r=0.97$), and further MRI scans were read by one or the other evaluator.

White-matter (WM) lesions were defined as areas in the WM, separate from the ventricles, that demonstrated prolonged T_2 relaxation times and appeared as areas of hyperintensity on the T_2 image. They were rated as periventricular if they were less than 1 cm from the ventricle (see Figs 1 and 2). They were assigned to an anatomic location (i.e. frontal, temporal, parietal, occipital) by reference to a standard neuroanatomic atlas (Roberts & Hanaway, 1970). All T_2 axial sections were screened for lesions. Coronal and sagittal images were used to corroborate and better define the location of lesions. All measurements were made by either BLM or CMM, who were independent and blind to diagnosis. To assess reliability in the measurement of WM lesions, we calculated intraclass correlations between the two raters by having them each read every fifth scan; inter-rater reliability was good ($r=0.83$).

Results

Approximately 250 patients over the age of 45 with psychosis were screened, and 24 met the entry criteria

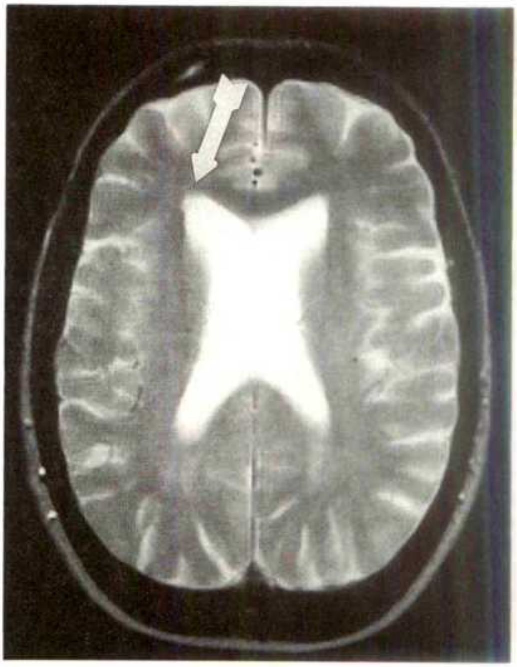

Fig. 1 T_2-weighted MRI scan of a normal elderly person, demonstrating a small periventricular rim of hyperintensity most prominent in the frontal and occipital regions.

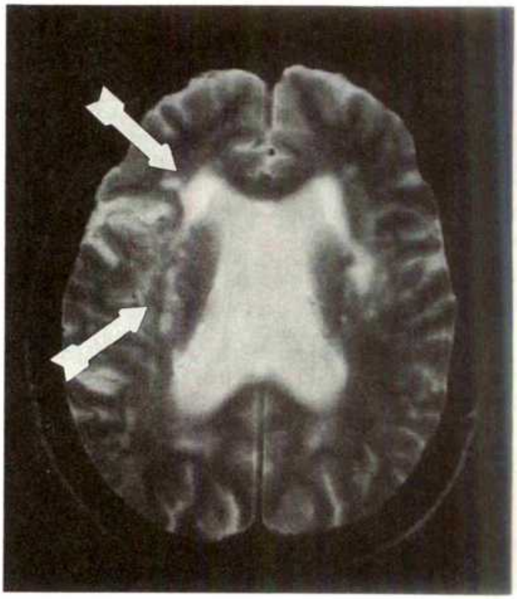

Fig. 2 T_2-weighted MRI scan in a patient with late-life psychosis, demonstrating large areas of hyperintensity in the periventricular, frontal, parietal and occipital regions.

described above. The major reasons for exclusion from the study included the onset of psychosis before age 45, a history of alcohol or drug abuse, non-fluency in English, or a failure to score 24 or better on the MMSE. An additional 14 subjects met the entry criteria but had a psychosis with a concurrent mood disorder and were analysed and reported separately (Lesser *et al*, 1990).

The mean age (s.d.) of the patients was 60.1 (10.1) years; 14 were women. Patients had an average 10.6 (3.3) years of education. Mean duration of illness was 19.9 (29.4) months. Fifteen were in-patients and nine were out-patients. Their mean BPRS score was 22.9 (8.5), and mean MMSE score was 28.0 (2.2). Ten patients were diagnosed as having a schizophrenic disorder, seven a delusional disorder, two a schizophreniform disorder, and five a psychosis, NOS. At the completion of the examinations, some diagnoses would have been changed to either organic delusional syndrome or organic hallucinosis; however, the classification presented here was based upon initial presentation of the patients. Seventeen of the patients experienced auditory hallucinations, and four had visual hallucinations. In no patient did we find a formal thought disorder (derailment, thought blocking, etc.).

Ninety controls were screened for inclusion and 72 were eventually studied. The major reasons for exclusion of controls were a past history of psychiatric or neurological disease or a recent history of alcohol abuse. The mean age for controls was 61.6 (10.0) years; mean time spent in education was 14.1 (2.8) years; 44 were women. The mean MMSE score for the controls was 29.5 (0.86), which was significantly higher than that for the patients ($t = 3.35$, $P < 0.003$).

Clinical evaluations

Three patients were unable to undergo MRI (one had a pacemaker; two became claustrophobic) and had only CT. A fourth patient previously reported (Lesser *et al*, 1987) was too large for either CT or MRI.

At the completion of the physical and neuroimaging evaluations, brain abnormalities were found in 10 of the 24 LLP patients (42%) and in 6 of the 72 of controls (8%) ($\chi^2 = 31.2$; $P < 0.001$, d.f. = 1). The brain abnormalities fell into four general categories: vascular lesions (cortical or WM infarctions) (6 of 24 patients; 5 of 72 controls), early primary degenerative dementia (by DSM-III-R criteria) (two patients, 1 control), tumour (one patient, no controls), and post-traumatic brain injury (one patient, no controls). These disease categories are described in more detail in the discussion. An additional four had abnormalities we have termed 'metabolic': psychoses related to cimetidine and ranitidine, congestive heart failure, codeine, and wytensin.

Measurements from MRI

The mean (s.d.) VBR was 10.6 (3.2) for the patients and 8.8 (3.6) for the controls. This difference approached but did not reach significance ($t = 1.76$, $P < 0.1$). There was wide variability in VBR for both groups and no indication of a subgroup of patients with increased VBR.

Table 1
Areas (cm^2; means and ranges) of white-matter lesions in patients and controls

	Patients		Controls	
Total	3.68	(0-23.6)	1.35	(0-15.9)
Periventricular	1.48	(0-8.07)	0.59	(0-11.5)
Temporal	0.44	(0-4.85)	0.07	(0-1.17)
Frontal	0.56	(0-6.25)	0.15	(0-2.98)
Occipital	0.82	(0-6.95)	0.20	(0-2.24)
Parietal	0.46	(0-5.55)	0.32	(0-3.76)

In the patient population, MRI showed a mixture of cortical and subcortical infarctions, with some patients demonstrating multiple large lesions. We did not find a preponderance of lesions in either hemisphere or in specific brain regions (Table 1). The overall distribution of the amount of WM lesions (not all counted as abnormalities) was very skewed for both groups because of the large numbers of subjects with no WM lesions (approximately a third of both groups); this prevented analysis by parametric statistical tests. When the non-parametric Kruskal–Wallis test was used no significant differences emerged between the groups. However, inspection of the data revealed that there was a subgroup of patients with a large amount of WM pathology.

The mean for temporal WM lesions was more than six times greater for patients than controls, and for both occipital and frontal lesions the patients had an approximately four times greater area of pathology. There were smaller differences between parietal and periventricular WM lesions. Because there are no established guidelines regarding the amount of WM lesion considered abnormal, we inspected the data and performed a χ^2 analysis, choosing cut-off points so that there were at least five subjects per cell. With cut-off points of 0, 0–4 cm^2, and >4 cm^2, seven patients (28%) and six controls (8%) had lesions <4 cm^2 ($\chi^2 = 4.84$, $P < 0.1$, d.f. = 2).

Neurological tests

The patient group had spent less time in education than the control group. Because education is correlated with performance on NP testing, we individually matched controls to patients on this variable (within three years), on age (within seven years) and sex. This was done without reference to the results of the clinical, MRI, or NP evaluations. Patients with no suitable control (e.g. those with sixth-grade education (age 11) or less) were excluded from this data set. This resulted in 21 matched pairs. We then conducted a series of matched-pairs *t*-tests between the patient group and their matched controls. After this, age and education were comparable for patients and controls (59.0 ± 8.8 years of age v. 59.1 ± 9.0 years; 11.3 ± 2.1 years of education v. 12.0 ± 1.6 years, respectively). The patients still had significantly lower scores on the MMSE (28.4 ± 2.0 v. 29.6 ± 0.7; $t = -2.39$; $P < 0.03$).

As seen in Table 2, performance on NP testing was consistently worse for the LLP patients, with significant differences on all but the WAIS–R digit span and the

Table 2
Matched-pair *t*-test comparisons between LLP patients and control subjects on NP tests (means ± s.d.)

	LLP	Controls	*t*	*P*
WAIS–R digit span, age-corrected scaled score	8.8 ± 3.5	10.1 ± 2.3	−1.31	0.21
WAIS–R full-scale IQ	84.5 ± 12.5	109.5 ± 12.6	−6.58	0.0001
WCST–categories	1.7 ± 2.3	4.2 ± 1.7	−4.18	0.0007
WCST–perseverative-response	64.6 ± 48.1	34.0 ± 25.8	2.13	0.057
FAS: total score	24.7 ± 9.4	39.6 ± 12.7	−3.87	0.0014
ACT: total score	29.9 ± 11.1	46.9 ± 7.8	−4.04	0.0012
Stroop: seconds	249.1 ± 125.7	133.1 ± 25.4	3.07	0.011
WMS–LM. % retention	45.6 ± 32.3	78.8 ± 25.0	−3.31	0.005
WMS–VR: % retention	40.6 ± 37.9	52.8 ± 22.3	−1.12	0.28
RMT–words: total score	36.9 ± 12.3	47.8 ± 1.9	−3.68	0.0022
RMT–faces: total score	33.2 ± 11.4	41.1 ± 3.6	−2.55	0.022
RO: copy score	23.9 ± 10.6	32.0 ± 5.8	−3.33	0.0045

WMS–VR. After correcting for multiple tests (requiring a $P < 0.004$ for significance at $P < 0.05$), differences on IQ, several of the frontal measures, and a verbal memory task still remained significant. Identical analyses were completed excluding the four patients with metabolic problems and their matched controls, with no change in results. Correlations were done with each NP test and the BPRS score; none were significant (all $r < 0.2$). When the patients were compared with the entire control group using analysis of covariance with education as the covariate, IQ remained significantly different. When education and IQ were used as covariates, the differences on NP testing paralleled those reported above for the matched-pair analyses.

Comparisons were made between the ten patients with structural brain disorders and the seven without such findings (excluding those with metabolic disorders). No differences were found between these two groups on age, educational level, IQ, or any of the NP measures. Scores from the five patients scoring lowest on each NP measure revealed nearly equal numbers of patients with and without brain abnormalities. Also, visual inspection of the protocols failed to reveal evidence of error types that were unique to one patient group.

Although the patients and controls did not receive extensive audiometry or ophthalmology tests, hearing and vision were tested during the neurological examination. None of the controls had serious visual or hearing impairment, while three of the patients had significant visual loss, one had serious hearing loss, and one patient had both. Those patients with visual loss were offered the use of a magnifying glass on NP testing. In those instances in which a sensory deficit might have contaminated a particular test finding, that specific test was not administered.

Discussion

The results of our comparison of patients with the onset of psychosis after aged 45 and healthy elderly controls demonstrate that in non-demented LLP patients with normal neurological examinations structural brain abnormalities are common (42%), and neuromedical illnesses (i.e. including 'metabolic' disorders) are even more common (58%). Neurologically silent vascular disease is present in a surprisingly high (25%) number of patients. Finally, intellectual, frontal-lobe, and verbal-memory deficits are prominent on NP testing.

Kay (1963) remarked that "late paraphrenia is a separate condition from the senile and arteriosclerotic dementias . . . and the main cause or causes must be looked for elsewhere than in the changes of either normal or pathological aging." He recognised, however, that a small minority, estimated as 5%, may have organic cerebral disease as an aetiological factor. Using modern technology, our findings (Miller & Lesser, 1988) and the findings of others (Leuchter & Spar, 1985; Holden, 1987) suggest that this figure may have underestimated the prevalence of brain abnormalities in patients with LLP.

In this study we evaluated only patients for whom there was no obvious organic cause for their LLP by excluding those with gross abnormalities on physical examination or MMSE, yet still found a high prevalence of brain lesions. Most of the patients were seen in the public health system and, as a group, tended to be of lower socioeconomic status. Also, one of the investigators in the study is a neurologist. Although these factors may have led to our seeing an increased prevalence of brain injury, the results appear robust, suggesting that even in a carefully screened group of patients, brain lesions are common with LLP. Because we excluded patients with known major medical or neurological disease, we believe the prevalence of brain abnormalities in an unselected group with LLP would be even higher.

Diseases seen concurrently with late-life psychosis

Although the patients had neither knowledge of a past stroke or focal neurological findings, the most common concurrent disease process was cerebrovascular disease evidenced on MRI as cortical and subcortical WM infarctions. In only two was there evidence suggesting neurological disease on physical examination (e.g. a gait disturbance). In one patient with a normal MRI, episodic transient ischaemic attacks involving the posterior cerebral arteries led to visual hallucinations and palinopsia, with ensuing delusions.

No single stroke syndrome predominated, although five had extensive infarction of subcortical WM with subcortical lacunar infarcts. This stroke pattern was first described by Binswanger in 1894, but until recently (Bradley *et al*, 1984; Brant-Zawadzki *et al*, 1985; Lotz *et al*, 1986; Hachinski *et al*, 1987) was thought to be uncommon. With the advent of CT and MRI, it has been demonstrated that WM injury occurs with increasing incidence, and is associated with ageing and hypertension, but its clinical significance is unclear (Goto *et al*, 1980; Steingart *et al*, 1987). It is possible that in some of the patients in our study the WM lesions were due to inflammatory brain disease such as is seen with multiple sclerosis (Edwards *et al*, 1986) or AIDS (de la Monte *et al*, 1987). However, no patient had a history suggestive of multiple sclerosis or AIDS, and the most significant risk factor seemed to be hypertension.

In our normal elderly population, multiple, but relatively small WM lesions and periventricular lesions were common (see Fig. 1), although extensive involvement of WM (see Fig. 2) was found much more often in patients than in controls. The areas where WM lesions showed the greatest difference between patients and controls were in the temporal, occipital and frontal areas. It is possible that the subcortical lesions affected the overlying cortex and played a role in the pathogenesis of the psychosis. Further study with a larger number of subjects will be necessary to explore this hypothesis.

Disease of WM may precipitate psychosis and depression. In previous reports, we described five patients in whom subfrontal WM infarction was associated with psychosis and a frontal syndrome (Miller *et al*, 1989) and significant WM disease in late-onset non-psychotic depressives (Lesser *et al*, 1990). Coffey *et al* (1989) reported that all 51 elderly patients with depression (some of whom were psychotic) studied had significant amounts of WM disease, which was more severe in those with a later onset of their depression. In another disease of WM, adult metachromatic leucodystrophy, psychosis often precedes the dementia syndrome (Skomer *et al*, 1983). WM lesions, in and of themselves, do not necessarily cause psychosis or depression, but their location, size, and the total brain volume affected, all may be important factors in producing psychiatric disorders in susceptible individuals.

Four of the 24 patients studied had metabolic or drug-induced psychosis. In all, the psychosis either disappeared or improved with the correction of medical conditions or discontinuation of medication. Although it is well known that psychosis can be induced or worsened by medication, in several of the patients in this study, the psychosis lasted for many months without other evidence for drug intoxication or delirium. The finding of drug-induced psychosis in non-delirious patients with LLP underscores the importance of reviewing all medication in these patients.

On cognitive testing, two patients had severe cognitive and memory problems, despite having MMSE scores in the normal range. As part of their broader evaluation, both had single-photon emission computerised tomography (SPECT) using an Elscint rotating gamma-counter scanner using technetium-labelled HMPAO, which showed temporal–parietal hypoperfusion typical of Alzheimer's disease (Johnson *et al*, 1987) in one, and bifrontal hypoperfusion seen with Pick's disease (Gustafson *et al*, 1987) in the other. These two patients thus appeared to be in the early stage of dementing illnesses with psychosis as the presenting clinical feature. Because psychosis is common in dementias (Cummings *et al*, 1987), assessment of LLP patients with functional imaging techniques may lead to an earlier diagnosis of the underlying disease.

Finally, one of the patients had a large tumour in the cerebellopontine angle, with compression of the brain-stem, and another had evidence on MRI for a traumatic brain injury. None of the controls had evidence for a brain mass or previous head trauma. Although this may have been an incidental finding, the data support a higher prevalence of brain tumour in patients in mental institutions (Galasko *et al*, 1988).

The role of neuropsychological testing

As a group, the patients had significantly lower IQ scores and performed more poorly than controls on NP tests assessing frontal-lobe functions and verbal memory. The severity of the psychosis (measured by the BPRS) did not correlate with the NP deficits. In 42 patients with 'late paraphrenia' (Hymas *et al*, 1989) NP deficits were present at onset and worsened over time. This occurred despite the attempt by the investigators to exclude patients with dementia.

We did not find statistically significant differences in NP performance between our patients with and without structural brain disease. Other investigations of chronic schizophrenics have not found a strong association between neurological status and NP performance (Klonoff *et al*, 1970; Goldstein & Halperin, 1977). The NP difficulties in our LLP patients are comparable with those documented in idiopathic schizophrenia (Kolb & Whishaw, 1983; Taylor & Adams, 1984), and suggest that similar neurophysiological mechanisms occur in both disorders. Neuroleptics might have influenced the NP scores in our patients. However, it is unlikely that neuroleptics alone caused the magnitude of large differences on NP tests, as all patients were tested in an alert and non-sedated state, and one would not expect neuroleptics to interfere differentially with performance on frontal-lobe and memory tasks, but spare other cognitive skills, especially attention. Also, the NP literature has not shown a consistent deleterious effect of neuroleptics on cognition in psychosis. In fact, neuroleptic use may actually increase performance on NP tests because of the lessening of psychotic symptoms (Heaton & Crowley, 1981).

Conclusion

In most of the patients we studied, there was nothing in the history, medical, or neurological examination that would have predicted the structural brain disease discovered, yet MRI detected brain abnormalities in over 40%. Although some abnormalities detected, such as post-traumatic injury and vascular infarction, may be untreatable, the information obtained is useful to clinicians. For example, there is evidence that in some patients with hypertension, WM pathology can be reversed or at least prevented from progressing (Hauser *et al*, 1988). In addition, psychosis associated with tumours and metabolic disorders may be completely reversed.

The issue of responsiveness to treatment was not systematically assessed. It is our impression that many of the patients studied were relatively resistant to traditional pharmacological and psychological treatment. However, some patients with brain injury do respond to treatment. In a patient with extensive WM lesions, dementia and a mood disorder, Joyce & Levy (1989) were able to improve the patient's mood disorder with antidepressants. It is possible that the underlying structural brain disease in our patients was a factor in their treatment resistance. This should be tested in a prospective treatment study.

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Mood Disorders in the Year after First Stroke

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An unselected community sample of 128 patients were studied over the 12 months after their first stroke, and compared with a control sample of subjects from the general population. Psychiatric status was assessed using the PSE and BDI. Symptoms of mood disorder were commoner in the stroke patients than the controls, but the differences were not substantial and had largely disappeared by 12 months. Psychiatric problems encountered included agoraphobia, social withdrawal, apathy and self-neglect, irritability and pathological emotionalism. While there was a high cumulative incidence of psychiatric disorder, little of it persisted: only two cases of major depression were present for the whole 12 months. We believe undue emphasis has been placed in the recent literature on major depression as a specific syndrome following stroke.

The possibility that patients who have had a stroke are particularly likely to develop psychiatric symptoms is of interest for a number of reasons. Firstly, those symptoms may in their own right form a part of the morbidity of stroke which is unrecognised because of the more dramatic physical component of the illness (Feibel *et al*, 1979). Secondly, mood disorder may add to the handicap caused by the physical effects of stroke, by impeding rehabilitation (Adams & Hurwitz, 1968; Robinson *et al*, 1984*a*; Sinyor *et al*, 1986*a*) or by impairing intellectual function (Robinson *et al*, 1986). Thirdly, of more theoretical interest, mood disorders after stroke have been claimed to have a different aetiology from those that accompany physical illnesses not involving the brain; in particular it has been claimed that depressive disorders are associated with the side and site of the cerebral lesion (Robinson *et al*, 1984*b*; Sinyor *et al*, 1986*b*).

However, there is no unequivocal information on the nature, frequency and natural history of the psychiatric sequelae of stroke. The main reasons for this are: selection bias towards in-patients and long-term out-patients; small numbers of subjects; the use of ill-defined selection criteria; and uncertainty about the validity of the methods used to measure psychiatric symptoms (House, 1987). Quoted rates of depressive disorder after stroke have therefore ranged from 18% to 61% (House, 1987).

From the point of view of sample bias, the major problem has been that most studies have been restricted to stroke patients admitted to hospital. Such patients may account for only 50–60% of the total population of stroke survivors and are not representative of the whole group; for example, they are more likely to have had a severe stroke, and to be living alone at the time of the stroke (Bamford *et al*, 1986). Hospital samples may be unrepresentative in terms of any ensuing psychiatric problems, since admission itself, regardless of its original cause, is a threatening experience (Brown & Harris, 1978). Studies of the prognosis of psychiatric disorder have also been compromised by irregular and incomplete follow-up in cohort studies, or by the use of convenience samples of those attending out-patient appointments in cross-sectional studies (Robinson & Price, 1982; Robinson *et al*, 1987).

The main diagnostic problem in previous studies has been reliance on informal clinical judgements, or on self-report inventories recording a range of affective, cognitive and somatic symptoms. Self-report inventories have a number of drawbacks: it is not always clear what contribution to the total score is made by symptoms of physical disease rather than emotional disturbance; the clinical severity of any psychopathology is difficult to judge; and it is not possible to give accurate descriptive diagnoses to those patients who are identified as cases. The studies of Robinson *et al* used an interview-based assessment to provide information upon which a psychiatric diagnosis was based, and therefore avoided some of these difficulties. However, the authors did not report emotional disorders such as anxiety, irritability or emotional lability, with the result that they have emphasised depression as the most important – or even the only – psychiatric disorder encountered after stroke. This conclusion has been encouraged by the use of 'post-stroke depression' as a quasi-diagnostic term (see e.g. Starkstein & Robinson, 1989).

The aims of the present study were to determine the range of psychiatric problems experienced by patients after a first stroke, their frequency and severity, and their course over the first year. We attempted to avoid the methodological problems outlined above in a number of ways. The patients studied were a consecutive series notified to the Oxfordshire Community Stroke Project who were diagnosed as having suffered a first stroke. The cohort was followed up on three occasions in the year after stroke, and assessed using standardised measures of psychiatic symptoms. A control group drawn from the same population was assessed using the same measures, so that we could compare results in the stroke patients with those in a general population sample with similar social characteristics.

This clinical description provides the background to other reports on the aetiology and consequences of mood disorder after stroke in the same group of patients.

Method

The Oxfordshire Community Stroke Project (OCSP) is a community-based research study of acute cerebrovascular disease in Oxfordshire, which recorded all cases from 50 general practitioners (in ten group practices) who worked in rural and urban areas, and who provided medical care to a population of approximately 105 000. Referred patients were seen by a research neurologist (MD or CW). In addition to a physical history and examination, assessments were made of cognitive function using Hodkinson's mental test score (Hodkinson, 1972) and a design-copying task; language was assessed on clinical testing. An estimate of disability was made using the Barthel index of Activities of Daily Living (ADL; Mahoney & Barthel, 1965); disability was assessed for the patient's function immediately before the stroke, as well as for the time of the neurological examination. Laboratory investigations were undertaken to identify known risk factors and to exclude rare causes of stroke. Final diagnosis was made at a clinical consensus meeting using a modification of the World Health Organization's definition of stroke (Aho *et al*, 1980). The OCSP has been described in detail by Bamford *et al* (1988).

Between 1 March and 30 November 1986, 116 patients were notified to the OCSP and were seen by a study neurologist and diagnosed as having suffered a first stroke. Ninety-five of these patients were alive one month after the stroke, and were first interviewed for the study described here between 31 March 1986 and 31 December 1986. Three patients alive at one month were excluded because they were *in extremis* and died within one week of the first interview. At six-month follow-up 91 of these 95 were alive, but one refused further interview and one had left the area. At one-year follow-up there were 86 survivors, of whom 84 were seen; one refused and one was uncontactable.

A further 33 patients were seen who had been notified to the project and had suffered their first stroke between November 1985 and March 1986. These patients – all those who had survived to the time of follow-up – were therefore interviewed for the first time at six months after their stroke. At one-year follow-up 31 of these patients were alive, and all were seen and interviewed.

Thus a total of 128 patients were seen at least once – 95 for the first time at one month and 33 for the first time at six months after the stroke. Of these 128 patients, 124 were alive and 122 were seen at six-month follow-up; and 115 out of 117 survivors were seen at one year. One retrospective exclusion not shown in these figures was a woman who was seen at one and six months, who died before one-year follow-up and was found to have a brain-stem glioma at post-morten examination.

A comparison group of patients was identified by taking a stratified random sample of the age–sex registers of four of the ten general practices from which the stroke patients were referred. The original sample ($n = 141$) was taken in 1986, but for practical reasons interviewing was undertaken during April–December 1987. At this time one patient had had a stroke and was excluded, eight had died, and four had left the area and were uncontactable, leaving 128, of whom 111 (87%) were interviewed (2 too demented and infirm, 4 thought interview would be too upsetting, 9 gave no reason, appearing simply uninterested, 2 had been recently bereaved and therefore refused).

Psychiatric assessment

Patients were usually seen at home; the only subjects seen in hospital were those who were in-patients at the time of follow-up. Mental state was assessed using the short version of the Present State Examination (PSE; Cooper *et al*, 1977) which was administered and rated by a trained interviewer (AH) during the course of an extended clinical interview. We did not modify either the standard form of questioning or the time covered (one month before interview), but we did make a minor change in rating criteria. The standard instruction for certain PSE items (e.g. tiredness) is that they should not be rated if they are understandable as a part of co-existing physical illness. We rated all symptoms without assumptions about their cause, so as not to prejudge this issue.

Patients completed the 21-item Beck Depression Inventory (BDI; Beck *et al* 1961) by themselves, unless problems with visual acuity or weakness meant that they needed assistance. Those who were unable to complete the BDI because of linguistic, praxic or other cognitive deficits did not have it administered to them. Cognitive function was assessed using the Mini-Mental State Exam (MMSE; Folstein *et al*, 1975), and the Frenchay Aphasia Screening Test (FAST; Enderby *et al*, 1987).

Subjects in the control group were interviewed on one occasion in their homes by a trained interviewer (LM) using the short version of the PSE, and they completed the BDI under the same conditions as the stroke patients. To maintain consistency in the use of the PSE, all LM's interviews were audiotaped and re-rated by AH; differences in ratings were then discussed at consensus meetings. A reliability exercise was undertaken on 12 audiotaped interviews (six tapes by AH, six tapes by LM).

Pathological emotionalism (sometimes called emotional lability) is a common problem after stroke, but there is no standard research definition of it. For the definition we adopted, patients were asked about three experiences:

an increase in episodes of crying (or more rarely laughing) since the stroke; the occurrence of such episodes with little or no warning; the inability to control these emotional episodes, so that they had occurred in front of other people. All three parts of the definition had to be met, and had to represent a change in the subject since the stroke. The context in which the emotional behaviour occurred was not taken as a defining part of the syndrome.

These assessments formed part of a wider study of psychiatric and social aspects of stroke, and other information was collected during the assessment interviews – about the patients' social status and relationships, and about their experiences of recent stress. These data will be reported in detail elsewhere.

Data analysis

Data from the PSE interview were analysed using the CATEGO/ID program (Wing *et al*, 1978), which produces a total symptom score and derives 14 clinical syndromes from the symptoms rated as present. The symptoms which each syndrome comprises are summarised in the legend to Fig. 3; further details are given by Wing *et al* (1974). The program also gives a measure of the severity of psychiatric symptoms, the Index of Definition (ID); a psychiatric diagnosis using the nomenclature of ICD–9 (World Health Organization, 1978) is given by the program to the 'cases' it identifies, that is, those with an ID of 5 or above.

In addition we made a diagnosis in each case according to Axis I of DSM–III (American Psychiatric Association, 1980), so that our results could be compared with those of Robinson *et al*. To do this we followed the method of the Johns Hopkins group, which was to add extra questions to the PSE interview, relating mainly to duration of mood symptoms outside as well as within the month before interview and to symptoms not usually included in the PSE such as recent weight gain or anorexia without weight loss. The PSE questions themselves were not modified.

Results from the BDI were expressed as total symptom scores and two subscores – the first 14 items were summed to score cognitive affective symptoms, and the remainder summed to score somatic symptoms. There is no universally agreed cut-off score on the BDI total to identify 'cases' of depression either in the general population or in medical patients (Beck, 1967; Moffick & Paykel, 1975; Nielsen & Williams, 1980; Cavanaugh, 1983, Cavanaugh *et al*, 1983; Gotham *et al*, 1986). We used several of the more commonly quoted thresholds to express our results so that comparison with other studies is possible. We have also reported a validation study comparing diagnoses reached using the BDI with those reached using the PSE (House *et al*, 1989*a*).

Other data were analysed using SPSS–X (Nie, 1983). Data from the two main psychiatric measures employed (PSE and BDI) were analysed using non-parametric statistics with two-tailed tests of hypotheses and a criterion of significance set at the 5% level.

Results

The social characteristics of the stroke patients and controls are shown in Table 1. More stroke patients were living in local-authority, health service or other professionally supervised accommodation (odds ratio 5.9, 95% confidence interval (CI) 1.9–18.1). Otherwise none of the differences was statistically significant.

On the indices of disability, the proportion of patients scoring less than the maximum on the Barthel ADL index was 18 of the 128 patients (14%) before stroke, and 58 of the 128 (45%) at assessment immediately after the stroke. Dysphasia defined by a FAST score of less than 20 was found in 19 of the 95 (20%) interviewed at one month. Cognitive impairment (MMSE score ⩽23) was present in 25 of 95 (26%) at one month, 26 of 122 (21%) at six months and 24 of 115 (21%) at 12 months.

Stroke subtype as determined by clinical presentation and computerised tomography was: probable or definite cerebral infarction in 104 (81%); primary intracerebral haemorrhage in 9 (7%); subarachnoid haemorrhage in 4 (3%); no scan, subtype uncertain in 11 (9%). At the time of the stroke 55 (43%) were being treated for hypertension, and 5 (4%) had diabetes managed by diet or oral hypoglycaemic agents. Only 48 of the 128 patients (38%) were admitted to hospital at the time of the stroke.

Scores on the BDI

The BDI was completed by 76 of 95 stroke patients (80%) at one month, by 107 of 122 (88%) at six months, and by 88 of 115 (77%) at one year. Of those who were able to complete the schedule at first interview 60 (79%) completed it on all three occasions. Of the 111 controls, 108 (97%) completed the BDI.

Table 1
Social characteristics of controls and patients at time of stroke

	Stroke patients ($n=128$)	Controls ($n=111$)
Age: years		
mean (s.d.)	71 2 (13.7)	69.6 (11.4)
range	18–96	30–86
Sex		
men/women	58/70	45/66
ratio	0.83:1	0.68.1
Social class[1]		
I	2 (1.5%)	4 (4%)
II	29 (22%)	33 (30%)
III	53 (40.5%)	38 (33%)
IV	36 (28%)	31 (28%)
V	8 (6%)	5 (5%)
Patient's living situation		
alone	26 (20%)	23 (21%)
family	84 (66%)	85 (76%)
non-family/institution	18 (14%)	3 (3%)

1. According to the Registrar General's classification.

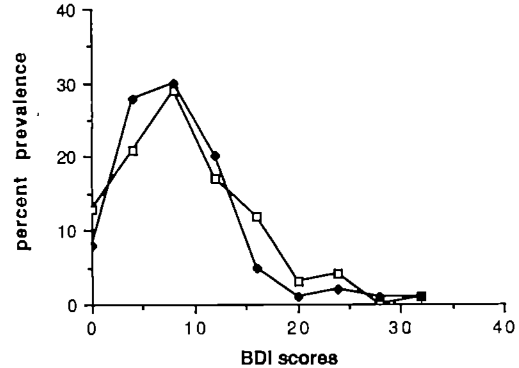

Fig. 1 Frequency distribution of BDI total scores for 76 stroke patients (1-month interview) (□—□) and 108 controls (■—■).

The frequency distribution of total BDI scores at one month and in the controls is shown in Fig. 1. BDI scores were not significantly different when the Mann–Whitney *U* test was used to compare control subjects with stroke patients at one month ($z=-0.54$, $P=0.59$), six months ($z=-0.62$, $P=0.53$) or 12 months ($z=-1.36$, $P=0.18$).

The frequency distribution of total scores on the BDI showed a shift to the left over the follow-up year, with fewer patients scoring in the higher range. Mean scores were 7.4 (s.d. 6.4) at 1 month, 7.2 (s.d. 5.6) at 6 months and 5.6 (s.d. 4.5) at 12 months. In fact for the 60 subjects who completed the BDI on all three occasions, the trend towards a reduction in symptom scores over the year did not quite reach statistical significance (Friedman two-way ANOVA, $\chi^2=5.66$, d.f. 2, $P=0.06$), but this may have been because patients in this subgroup initially had less severe symptoms than the total sample (of the original 76 BDI respondents at one month, 32% had BDI scores of 10 or over, compared with 23% of the subgroup of 60 who completed three interviews). Comparing the scores of the subjects who completed a BDI on at least *two* occasions using the Wilcoxon matched-pairs signed-rank test, the reduction was significant for scores at one and six months ($n=69$, $z=-2.74$, $P=0.006$) and at one and 12 months ($n=62$, $z=-2.30$, $P=0.02$); the Spearman rank-order coefficients of correlation for these same patients were: 1 month v. 6 months ($n=69$) $r=0.65$, 1 month v. 12 months ($n=62$) $r=0.58$, 6 months v. 12 months ($n=84$) $r=0.70$ (all significant, $P<0.001$) (i.e. patients with high scores at one interview still had high scores subsequently).

The relative contribution of somatic and cognitive affective subscores to the overall BDI score for the stroke and control subjects is shown in Fig. 2. For the stroke patients, the decline in scores for somatic symptoms over 12 months was significant (Friedman two-way ANOVA $\chi^2=10.21$, d.f. 2, $P=0.006$) but the change in scores for cognitive affective symptoms was not (Friedman two-way ANOVA, $\chi^2=0.33$, $P>0.1$). As with the total BDI scores, there were no significant differences on either of the subscores between the stroke patients at any interview and the controls, with one exception: at 12 months the score for somatic symptoms was lower in the stroke patients than in the controls ($z=-2.59$, $P=0.01$). However, this result comes from one of six tests applied to BDI subscores, and the overall *P* value can only be claimed as less than 0.06 – it is of doubtful clinical significance.

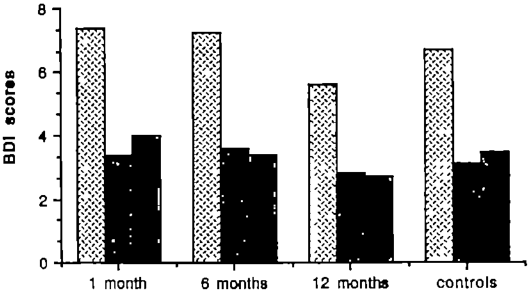

Fig. 2 Mean BDI total scores (▨) and mean scores on the cognitive affective (■) and somatic (▨) sections, for 76 stroke patients at 1 month, 107 at 6 months, and 88 at 12 months after stroke, and for 108 controls.

The frequency with which individual BDI items were rated positively is shown in Table 2. Some interesting observations emerge from these data: (a) the decrease in total symptom score in the year after stroke was accounted for by a *general* decline in symptoms; (b) certain symptoms

Table 2
Prevalence of BDI items after stroke and in controls

	Stroke patients			Controls
	1 month ($n=76$)	6 months ($n=107$)	12 months ($n=88$)	($n=108$)
Sadness	19 (25%)	31 (29%)	19 (22%)	18 (17%)
Pessimism	20 (26%)	25 (23%)	11 (13%)	13 (12%)
Sense of failure	7 (9%)	9 (8%)	8 (9%)	13 (12%)
Lack of satisfaction	29 (38%)	43 (40%)	34 (39%)	41 (38%)
Guilt	6 (8%)	10 (9%)	3 (3%)	10 (9%)
Sense of punishment	5 (7%)	5 (5%)	1 (1%)	8 (7%)
Self-hate	15 (20%)	25 (23%)	16 (18%)	21 (19%)
Self-accusation	13 (17%)	22 (21%)	18 (20%)	29 (27%)
Suicidal ideas	2 (3%)	3 (3%)	4 (5%)	2 (2%)
Crying	13 (17%)	29 (27%)	18 (20%)	29 (27%)
Irritability	30 (40%)	43 (40%)	30 (34%)	41 (38%)
Social withdrawal	13 (17%)	14 (13%)	12 (14%)	17 (16%)
Indecisiveness	23 (30%)	37 (35%)	24 (27%)	27 (25%)
Body image	8 (11%)	13 (12%)	9 (10%)	22 (20%)
Work inhibition	52 (68%)	72 (67%)	56 (64%)	58 (53%)
Sleep disturbance	34 (45%)	41 (38%)	28 (32%)	54 (50%)
Fatiguability	54 (71%)	76 (71%)	59 (67%)	73 (67%)
Loss of appetite	17 (22%)	22 (21%)	9 (10%)	17 (16%)
Weight loss	17 (22%)	20 (19%)	9 (10%)	9 (8%)
Somatic preoccupation	21 (28%)	24 (22%)	17 (19%)	18 (17%)
Loss of libido	20 (26%)	19 (18%)	16 (18%)	38 (35%)

of depressive thinking – guilty ideas, a sense of being punished, suicidal ideas, a sense of failure – were uncommon at all interviews in the stroke patients; (c) some items were commonly reported by the controls and were no more frequent (or even less frequent) in the stroke patients (e.g. loss of libido, weight loss, sleep disturbance).

Psychiatric cases defined on BDI criteria

The proportion of depressed cases identified using three different BDI cut-off points are given in Table 3. Although there were clear trends for patients seen one and six months after stroke to be more likely than controls to be cases on each BDI criterion, only at six months, using a score of $\geqslant 10$, did this reach statistical significance ($\chi^2 = 4.2$, d.f. 2, $P = 0.04$).

Despite relatively high correlations between BDI scores at different follow-up interviews, the cases identified on each of the three interviews in the follow-up year were not the same individuals. For example, of the 14 cases who scored $\geqslant 13$ at one month and completed the BDI at six months, only six still scored $\geqslant 13$, while the other ten patients who scored $\geqslant 13$ at six months had not met this criterion at one month.

For the total group of 128 stroke patients, 50 (39%) were identified as cases at some time during the year using a BDI cut-off score $\geqslant 10$, and 26 (20%) using a cut-off score $\geqslant 13$. However, very few patients remained cases throughout the year: of the 60 who completed the BDI three times in the year, only four people had scores $\geqslant 10$ on all three occasions, and only two had BDI scores $\geqslant 13$ on all three occasions. These figures are almost certainly underestimates of psychiatric morbidity because of the subjects who did not complete the BDI on every occasion, but a similar pattern was seen with PSE-defined cases where the response rate was higher.

Table 3
Prevalence of psychiatric disorder according to different BDI criteria and using PSE/ID method of case definition, for stroke patients and controls

	BDI total score			PSE/index of definition		
	$\geqslant 10$	$\geqslant 13$	$\geqslant 17$	1–3	4	5+
Stroke patients						
1 month (BDI $n = 76$, PSE $n = 89$)	24 (32%)	15 (20%)	6 (8%)	48 (54%)	24 (27%)	17 (19%)
6 months (BDI $n = 107$, PSE $n = 119$)	35 (32%)	16 (15%)	6 (6%)	70 (60%)	26 (22%)	23 (19%)
12 months (BDI $n = 88$, PSE $n = 112$)	14 (16%)	7 (8%)	1 (1%)	80 (71%)	20 (18%)	12 (11%)
Controls (BDI $n = 108$, PSE $n = 109$)	22 (20%)	11 (10%)	5 (5%)	88 (81%)	9 (8%)	12 (11%)

The Present State Examination

In the stroke group 89 (94%) of the 95 patients interviewed at one month were able to provide ratable answers during the PSE interview, as were 119 of 122 (98%) at six months and 112 of 115 (97%) at 12 months. In the control group 109 (98%) were interviewed.

For the 12 tapes which were independently rated in the reliability study, mean total symptom scores (s.d.) were 12.5 (10.3) (AH rating) and 12.6 (9.5) (LM rating), which was not a significant difference (Wilcoxon matched-pairs signed-ranks test) (Spearman's correlation for total scores $r = 0.99$). Seven cases (ID 5+) were identified with 100% agreement between raters; there was complete agreement on ID in 6 of 7 cases and agreement within one ID category in 11 of the 12 subjects.

The frequency distribution of PSE total symptom scores was very similar to that of the BDI scores, with a positive skew and stroke cases shifted to the right when compared with controls. Mean total score in the stroke patients seen at one month was 6.5 (s.d. 5.6) and in the controls 3.4 (s.d. 2.8). The PSE total scores in the controls were significantly lower using the Mann–Whitney *U* test to compare them with stroke patients at one month ($z = -4.45$, $P < 0.0001$), six months ($z = -3.72$, $P = 0.0002$) and 12 months ($z = -2.38$, $P = 0.02$).

The frequency distribution of total scores on the PSE showed a shift over the year, with fewer patients scoring in the upper ranges. Mean total PSE scores were 6.5 (s.d. 5.6) at one month, 5.5 (s.d. 4.5) at six months, and 4.7 (s.d. 4.3) at 12 months. For the 78 patients who completed the PSE on all three occasions, the reduction in total score was significant (Friedman two-way ANOVA, $\chi^2 = 13.58$, d.f. 2, $P = 0.001$).

As with the BDI, there was a moderately strong association between scores at the different interviews. For patients who completed the PSE on at least two occasions, the Spearman rank-order correlation coefficients were: 1 month v. 6 months ($n = 83$) $r = 0.62$; 1 month v. 12 months ($n = 80$) $r = 0.65$; 6 months v. 12 months ($n = 110$) $r = 0.66$ (all significant, $P < 0.001$).

The frequency of the various syndromes defined by CATEGO/ID in the stroke patients seen at one month and in the controls is illustrated in Fig. 3. The difference in total scores between the groups was accounted for by a greater prevalence in the stroke patients of nearly all PSE syndromes, with the exceptions of special features of depression (ED), situational anxiety (SA), and tension (TE).

There was a general decline in the frequency of cognitive, affective and somatic syndromes over the follow-up year. The only syndromes still found in more than half the patients by the end of the first year were worry (WO) and lack of energy (LE); by comparison there had, for example, been a sharp decline in the frequency of the commonest depressive syndrome (SD). Situational anxiety (SA) – which in this study was entirely agoraphobia – persisted in those who had it at the start of the year, as did the related (non-phobic) symptom of social withdrawal, which had a prevalence of 15–16% on all three occasions. These latter symptoms were associated with worry about changes in functional ability, fear of looking conspicuous due to

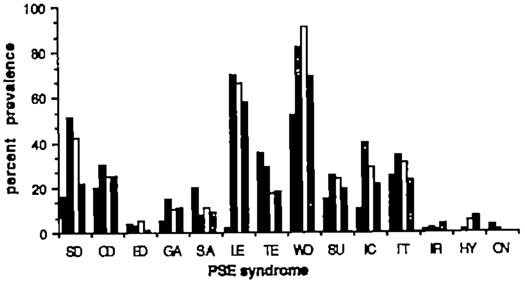

Fig. 3 PSE syndrome profiles for stroke patients seen at one ($n=89$, ■), six ($n=119$, □), and 12 ($n=112$, ▨) months after stroke and for 109 controls (■). SD, *simple depression* (inefficient thinking, depressed mood; hopelessness; suicidal plans or acts; depression on examination); OD, *other symptoms of depression* (morning depression; loss of appetite; early waking; loss of libido; pre-menstrual exacerbation); ED, *special features of depression* (self-depreciation; guilty ideas of reference; guilt, dulled perception; lost affect); GA, *general anxiety* (anxiety; panic attacks; anxiety on examination); SA, *situational anxiety* (situational anxiety; specific phobias; anxiety avoidance); LE, *lack of energy* (subjective anergia); TE, *tension* (tension pains; muscular tension; restlessness); WO, *worry* (worrying; tiredness; nervous tension; delayed sleep); SU, *social unease* (anxiety on meeting people; social withdrawal; lack of self-confidence); IC, *loss of interest and concentration* (poor concentration; loss of interest); IT, *irritability* (irritability; hostile irritability); IR, *ideas of reference* (ideas of reference); HY, *hypochondriasis* (hypochondriasis); ON, *obsessional neurosis* (checking and repeating, cleanliness and rituals; obsessional ideas and rumination).

hemiparesis or facial asymmetry, a desire to avoid friends and acquaintances because of uncertainty about their response to the subject, and a fear of recurrence being brought on by physical activity. Such concerns rarely reached sufficient severity to rate as ideas of reference (IR) or hypochondriasis (HY). Irritability declined in frequency from 36% at one month to 21% at 12 months. This change was entirely in the group who felt irritable but kept their feelings to themselves; the proportion who expressed irritability to others stayed constant at 12%.

PSE-defined cases

The proportion of stroke patients identified as cases (ID $\geqslant$ 5) at each interview is shown in Table 3, which also shows the proportion who had specific symptoms (i.e. depression or anxiety) without reaching the cut-off point. The apparent decline in the proportion of those at ID $\geqslant$ 5 was not enough to reach statistical significance with our sample size (Cochran's $Q=5.05$, d.f. 2, $P=0.08$) for those who were interviewed on all three occasions. This was not due to the subgroup who completed the PSE on all three occasions having less severe symptoms than the total population to begin with, since 15 of the 78 (19%) who did so also scored at ID $\geqslant$ 5 at the one-month interview. Although the proportion of cases did not change greatly, it was – as with the BDI – not the same individuals who were identified as cases at each interview. For example, of the 15 initial cases interviewed three times, at six months only seven were still cases – the other eight cases at six months had not been cases at one month.

Table 4
Principal diagnoses according to CATEGO ICD-9) and Axis I (DSM-III) for the stroke patients and controls

	Stroke patients			Controls ($n=109$)
	1 month ($n=89$)	6 months ($n=119$)	12 months ($n=112$)	
CATEGO diagnosis (ICD-9 categories)				
anxiety neurosis	3 (3.5%)	3 (2.5%)	3 (3%)	2 (1.5%)
phobic neurosis	3 (3.5%)	4 (3.5%)	3 (3%)	1 (1%)
depressive neurosis	11 (12%)	16 (13%)	6 (5%)	9 (7.5%)
DSM-III diagnosis				
major depression	10 (11%)	12 (9%)	6 (5%)	9 (7 5%)
dysthymic disorder	1 (1%)	3 (3%)	5 (4%)	1 (1%)
adjustment disorder (depressed)	11 (11%)	10 (8%)	6 (5%)	0
atypical affective disorder	0	1 (1%)	0	0
agoraphobia ± panic	2 (2%)	3 (3%)	2 (2%)	1 (1%)
generalised anxiety	1 (1%)	1 (1%)	0	1 (1%)
adjustment disorder (anxious)	4 (4%)	4 (3%)	5 (4%)	1 (1%)
adjustment disorder (mixed) disorder of behaviour and emotions)	7 (8%)	3 (3%)	1 (1%)	0
conversion disorder	1	1	0	0
Totals	37 (42%)	38 (32%)	25 (21%)	13 (12%)

For the total group of 128 stroke patients, 28 (22%) were identified as cases of psychiatric disorder according to PSE/ID criteria at some time in the year following their stroke. In contrast to this high cumulative incidence, the persistence of 'caseness' throughout the year was uncommon; only 3 of 78 (4%) patients who completed the PSE on all three occasions during the year scored five or more on ID on every occasion.

Psychiatric diagnosis and pathological emotionalism

The ICD-9 diagnoses given by CATEGO to the stroke patients who scored at ID $\geqslant$ 5 at each interview are shown in Table 4, which also shows the principal diagnoses on DSM-III Axis I. Because the criteria for a diagnosis on Axis I are less stringent than those applied by the CATEGO program, the overall rates of psychiatric disorder obtained by using the former method were higher. The rates of major depression and anxiety disorders combined were however very similar to those obtained for mood disorders by the other diagnostic criteria employed in this study. Again, the persistence of disorder throughout the year was much less common. Only 8 of 78 (10%) of those interviewed on all three occasions in the year received a DSM-III diagnosis on each occasion. Major depression persisted throughout the year in two (3%) of these 78 patients.

In the control subjects, 12 of the 109 (11%) who were able to complete the PSE interview were rated at ID$\geqslant$5 (see Table 3). Nine received a CATEGO diagnosis of depressive neurosis, two had a diagnosis of anxiety neurosis, and one had phobic neurosis (agoraphobia). All the depressed cases fulfilled the criteria for a diagnosis of major depression on Axis I.

Using the definition given under Method, pathological emotionalism was experienced by 13 of 89 (15%) stroke patients at one month, 25 of 119 (21%) at six months, and 13 of 112 (12%) at 12 months. Two patients reported episodes of pathological laughter in the first six months. We have described elsewhere our findings on the associations between emotionalism and other aspects of the patients' psychological and neurological state (House *et al*, 1989*b*).

A common complaint of carers was that the stroke patients were apathetic, and that this was a change – at least in degree – from their previous personality. This observation, which was difficult to quantify, was seen as a major problem by some carers and a minor annoyance by others. It was reported in 13 (11%) at six months, and only three (3%) at 12 months. An unrelated problem (which is often confused with this social indifference) was the tendency of some patients to downplay or deny the existence of disability or handicap arising from paralysis. We encountered only one fully developed example of denial (House & Hodges, 1988) and two cases in which there was partial denial of handicap as a transient phenomenon during the acute phase of recovery from severe hemiparesis.

Eight patients were identified as having had definite psychiatric disorders in the 12 months before their stroke; that is, they either described a clear onset of major mood symptoms antedating the stroke and persisting to the time of the first interview, or they were receiving psychiatric treatment at the time of the stroke. Of the 17 cases identified by CATEGO/ID at one month, five had psychiatric disorders preceding the stroke. On DSM-III criteria all five were diagnosed as having major depression at one month.

Patients with psychiatric disorders which persisted throughout the follow-up year were particularly likely to have had psychiatric problems in the year before stroke. Two of the three persistent cases identified on CATEGO/ID criteria belonged to this group. Of the eight patients who were interview on three occasions and received a DSM-III diagnosis each time, four had their onset in the 12 months before stroke; a further three had a history of psychiatric problems but had not been treated in the preceding year and did not describe case levels of symptoms antedating the stroke. Of the two cases who were diagnosed as having major depression on all three interviews, one had a chronic disorder and was a psychiatric out-patient after in-patient treatment; the other was seeing her general practitioner at the time of the stroke, with anxiety symptoms and (at that time undisclosed) alcohol abuse.

Discussion

This was a community-based study of sufferers of a first stroke, many of whom were quite mildly disabled (one indication being the low hospital admission rate at the time of stroke). This almost certainly accounts for some of the differences between our results and those of other studies, which have used only cases referred and admitted to hospital and have included patients with recurrent strokes at entry. Refusals among the controls did not, as far as we could judge, bias the results substantially.

The measures of psychopathology employed also deserve comment. One problem with the BDI is that a substantial minority of stroke patients cannot complete it. We chose not to administer the BDI by interview to avoid the problem of spuriously high agreement between different measures, as might be obtained by a single interviewer completing several different inventories with a subject at one interview. Our experience is that most of those who cannot complete the BDI unassisted are unable to follow the forced-choice format when it is read out. Our response rates were as high as those of others who have used self-report inventories, so it is unlikely that serious inaccuracy resulted from our adoption of this procedure. However, our BDI results do therefore refer to a population able to complete the measure themselves. There was no suggestion of bimodality in the distribution of BDI scores, so that any assignation of 'caseness' on the basis of them is bound to be arbitrary. This suggests that while schedules such as the BDI might provide reasonable approximations to the prevalence of depression in a population, they are likely to be a relatively inaccurate way of defining individual cases.

In using the shorter version of the PSE we did not modify the standard format of the interview but we did adopt the position, taken by others (Feldman *et al*, 1987), that all symptoms were rated without assumptions about their cause. The same convention was adopted with the control group, but this difference – which in practice has an effect on the prevalence only of certain non-specific symptoms – needs to be born in mind if the results are to be compared with those of surveys conducted using another convention.

The aims of the study were to determine the nature, frequency and prognosis of psychiatric disorder in the year after stroke. What were the findings and how do they compare with those of others whose work prompted our own research?

It is clear that after stroke a wide variety of emotional disorders may develop. As in other settings, anxiety may coexist with or occur independently of depression. This is important because agoraphobia appeared to be particularly persistent. Other less clearly delineated types of adjustment reaction included irritability, social withdrawal, excessive concern about dementia or recurrence, and a range of 'non-specific' physical symptoms. These syndromes, which accounted for a substantial part of the psychiatric morbidity after stroke, are often neglected because there is no satisfactory nomenclature for them. It is our impression that they constitute much of what is often referred to as personality change in stroke patients, which is unfortunate because that phrase carries with it an implicit assumption of irreversibility.

The findings on pathological emotionalism highlight the frequency of this common but little understood phenomenon (House *et al*, 1989*b*). Another common syndrome that is often thought of as an inevitable consequence of brain damage for some people is that of morbid apathy or indifference (Heilman *et al*, 1985). In this study it was predominantly a transient phenomenon which had largely resolved by the end of the year, suggesting that it too might be seen as a form of adjustment reaction or at the least as a non-permanent sequel of stroke.

'Depression' is often used as a catch-all term for the emotional problems discussed above, but the experience of these other states for patient and carer – and the best approach to management – may well not be the same as for depressive disorders. It is for that reason that we have emphasised their existence. From the point of view of depressive disorders themselves, it has been argued that in stroke patients major depression is qualitatively different from other depressive syndromes found in stroke patients (Starkstein & Robinson, 1989). As noted above, scores on the BDI and the PSE showed no evidence of a bimodal distribution, which would suggest that diagnosable depression, and psychiatric 'caseness' in general, is mainly a function of symptom severity and frequency rather than specific content. Studies of general medical populations have shown very similar distributions of symptom scores to our own, and similarly low rates of symptoms such as guilt, ideas of being punished and suicidal plans (Moffic & Paykel, 1975; Cavanaugh, 1983*a*). There was no evidence from this study that the depression found in stroke survivors differs from that found in patients with other physical illnesses.

What about the frequency with which psychiatric disorder was found? For the controls, comparison with other community surveys is difficult because of the variability of the results obtained elsewhere (see Swartz & Blazer (1986) for discussion). Bearing that in mind, rates for 'dysphoric symptoms' as measured by the BDI, for PSE cases and for major depression, are of the same order as have been reported by others. Especially in the early months after their stroke, patients had levels of psychiatric symptoms which were higher than those of the controls. However, the rates were very much lower than some that have been reported in stroke patients or in other physically ill populations. What is the explanation for the difference?

One factor may be the population studied: our patients – who were recovering from their first stroke – were less disabled, and less likely to have been admitted to hospital or to be suffering from recurrent stroke than others who have been reported. Also they were less socially deprived than some, particularly the patients studied by Robinson and colleagues. Oxfordshire is an affluent county in the south of England with high levels of employment, and the population studied by the OCSP lives mostly in villages and small towns which have few of the social problems of large industrial cities. Two other studies conducted in the same area have also shown low rates of psychiatric disorder among the elderly on general medical wards (Feldman *et al*, 1987), and in long-term survivors of stroke (Sharpe *et al*, 1991).

The second likely explanation lies in the measures and definitions of psychiatric disorder employed. Questionnaires are difficult to interpret for the reasons already discussed. In addition, in the present study the BDI may have underestimated rates because of our decision to adhere to self-administration as the method for completion. This conclusion would be supported by the finding that those who were well enough to complete the form on all three occasions had less severe symptoms than those who could not do so.

By contrast, a standardised clinical interview should enable an orthodox psychiatric diagnosis to be made, allowing a common nomenclature to be employed across studies. Only the study of Robinson *et al* used a similar method to ours but comparison is difficult, in part because they modified the PSE so that CATEGO/ID analysis was not undertaken. There are two reasons why their method (rather than study population) might have led to a much higher estimate of rates of depression than we found. Firstly, they apparently altered the time constraints normally used as part of the DSM–III diagnostic criteria; for example they use 'dysthymic depression' to refer to all depressive episodes which do not meet the criteria for major depression. They have also changed the time criterion for major depression itself, which could lead to a serious overestimation of rates by inclusion of patients with insufficiently persistent disorders. Thus patients interviewed within seven days or less of stroke were described as having "symptom clusters associated with major depression", and were given that diagnostic label (Robinson *et al*, 1983). Secondly, their failure to mention affective syndromes other than depression raises the possibility that disorders included in their depressive categories have been given alternative designations in our study; this may apply particularly to the non-depressive adjustment disorders and to pathological emotionalism.

The third part of the present study involved an investigation of the course of psychiatric disorder over the first year after stroke. The finding that levels of psychiatric symptoms declined over the year is at odds with claims that mood disorders after stroke are unusually persistent (Robinson & Price, 1982; Robinson *et al*, 1987; Wade *et al*, 1987). However, of the reports in which these claims are made, only one involved a cohort study with acceptable rates of follow-up and that used a self-report inventory with an arbitrarily chosen threshold score (Wade *et al*, 1987). Much work in this field has been characterised by reliance on grouped data where (as in our study) total symptom scores correlated quite highly across time, and changes in population mean scores or the proportion scoring above a given cut-off score were not great. Such observations obscure the fact that the individuals who are high scorers/cases may not be the same on each occasion. In the present study there was a high cumulative incidence of cases, particularly in the first six months, but very little diagnosable psychiatric disorder which persisted throughout the study year. In fact, the small proportion of persistent cases were mainly those who had psychiatric problems before their stroke, who had continued with psychiatric disorder into the follow-up year. The most vigorous proponents of an alternative view have been Robinson *et al*, whose claim is that mood disorder starts early after stroke and persists in a majority for one or two years. They have based this argument on studies of long-term hospital out-patient attenders and on irregular and incomplete follow-up of selected cohorts, and their results are likely to be seriously biased as a consequence.

In summary, depression is not the only mood disorder to appear as a sequel to stroke, and when it does occur it is not qualitatively different from the depression reported in association with other medical conditions. Insufficient attention has been paid in the recent literature to other persistent and potentially disabling conditions such as agoraphobia and pathological emotionalism, or to the so-called 'non-specific' symptoms such as lack of energy and tension which were so common in the patients we interviewed. The frequency of depressive disorders after stroke and their persistence have been overestimated. For example, there are no reports of substantial studies comparing rates of mood disorder after stroke to rates in the general population using a case-control design. For these reasons the phrase 'post-stroke depression' is misleading, since it implies the existence of a discrete, particularly common and in some way specific clinical syndrome, and we suggest that its use as a descriptive term is discontinued.

Nonetheless, we have found that a wide variety of emotional disorders are commoner in the year after stroke than they are in the general population. Their cause or clinical picture could be specifically associated with the cerebral lesion in stroke, or their aetiology could be the same as for those mood disorders which arise in medical patients without brain disease. That is, they may be related to the threat inherent in physical illness and hospital contact, the associated physical disability and social handicap, and stressful life experiences unrelated to the subject's illness. In future papers we will be reporting our findings on the aetiology of mood disorders after stroke, which we have examined in this unique community-based psychiatric study of stroke survivors.

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The Impact of a Child Psychiatry Liaison Service on Patterns of Referral

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A retrospective study of referrals to a child psychiatry liaison service was carried out in order to assess the impact of the reorganisation of the service. A total of 55% of referrals were of in-patients on the paediatric ward; other in-patients made up 12.5% Of all referrals, 67% were from paediatricians Of the children who had not harmed themselves, most were referred either for help with the management of physical illness or for investigation of a non-organic physical complaint. The main finding was that liaison referrals increased significantly in contrast to both the total number of referrals and the number of cases of deliberate self-harm.

The psychological sequelae of both illness and admission to hospital in children have been well documented (Prugh *et al*, 1953; Mrazek, 1986). However, it is only in recent years that the concept of liaison child psychiatry has begun to evolve in the UK (Taylor, 1986). As in adult liaison psychiatry (Lloyd, 1980), services in which the psychiatrist is integrally involved in the management of patients presenting with physical symptoms have probably not been developed in the UK to the same extent as in the US.

Stocking *et al* (1970) and Awad & Poznanski (1975) have confirmed high rates of psychopathology in children admitted to hospital. The latter found that patients were more likely to be referred to child psychiatrists when the symptoms were of unknown aetiology, depression was suspected or behaviour problems arose on the ward. Stocking *et al* studied the circumstances in which the paediatrician was likely to overlook emotional disorder. These included the presence of an acute treatable medical condition, especially in younger children.

In 1973, Monnelly *et al* drew attention to the paucity of research in this area, and in particular to the lack of systematic studies of psychiatric consultations in paediatric hospitals. Since then, analyses of the type of referrals to child psychiatrists and of services offered by such psychiatrists to their paediatric colleagues have been carried out at a number of centres (e.g. Wrate & Kolvin, 1978). Trends in referral to adult liaison services have been studied by Lipowski & Wolston (1981) and Brown & Cooper (1987).

Naylor & Mattson (1973) and Jellinek *et al* (1981), among others, have described the development of child psychiatry liaison services. This has led to the assessment of the efficacy of therapeutic interventions in physical illnesses. Lask & Matthew (1979) demonstrated that family therapy is not only useful in alleviating the stress related to a physical illness (asthma) but may also have an impact on the course of the disease. Systemic thinking has aided the understanding of processes involved and contributed to the management of organic and non-organic physical complaints. The value of this approach has been highlighted by Bingley *et al* (1980).

The Royal Free Hospital in north London is a district general hospital serving a population of approximately 100 000 people. As well as providing a general paediatric service, it is also the supraregional centre for bone-marrow transplantation and for the management of end-stage renal failure in children.

There has been a child psychiatry service based in the hospital since 1971, but in 1984 the newly appointed consultant child psychiatrist undertook responsibility for the co-ordination of psychosocial services for children in the hospital. A decision was made at that time to improve the child psychiatry liaison service, one aspect of which has been described by Jaffa (1988).

This paper describes a study evaluating the impact of the decision. Its aims were:

(a) to describe 'liaison' patients in demographic and diagnostic terms
(b) to describe the source and nature of referrals
(c) to examine changes in patterns of referral over a three-year period.

Method

A retrospective case-record study was carried out on all patients referred by other hospital departments to the department of child psychiatry in six alternate three-month periods from mid-1984 to mid-1987. Resources did not

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Based on a paper presented to the Annual Residential Meeting of the Royal College of Psychiatrists' Child and Adolescent Psychiatry Section, Oxford, September, 1987

Table 1
Reasons for referrals of all patients ($n = 125$) except those with deliberate self-harm ($n = 34$)

	Autumn '84	Spring '85	Autumn '85	Spring '86	Autumn '86	Spring '87
Physical illness	12 (57%)	9 (50%)	3 (20%)	6 (43%)	18 (67%)	15 (50%)
Physical symptoms (non-organic)	2 (9%)	3 (17%)	6 (40%)	0 (0%)	3 (11%)	4 (13%)
Habit disorders	4 (19%)	4 (22%)	1 (7%)	2 (14%)	3 (11%)	7 (23%)
Childcare problems	0 (0%)	2 (11%)	4 (27%)	4 (29%)	3 (11%)	3 (10%)
Behaviour problems	3 (16%)	0 (0%)	1 (7%)	0 (0%)	0 (0%)	1 (4%)
Psychosis	0 (0%)	0 (0%)	0 (0%)	2 (14%)	0 (0%)	0 (0%)

permit us to look at the full population. The following data were recorded: age, sex, social class and ethnicity of referred patients; source and nature of referral; presence of self-poisoning or injury; and diagnosis and service given. Comments about family functioning as recorded in the case notes by the professionals involved were also noted.

In order to compare the proportions of liaison referrals in each year, statistical analysis was carried out using linear logistic analysis.

Results

The mean age of referred patients was calculated for the total sample ($n = 159$) and for each time period. There was no significant difference in age between periods, although two adults were index patients during the last. Excluding these two patients, the mean age was 10.4 years for all patients, and 9.28 years if those with self-poisoning or injury were excluded.

The ratio of female:male patients did not vary significantly with time; the overall ratio was 1.5:1. Approximately a third (32%) of patients referred were either non-British or belonged to a family of non-British origin. Information on social class was least reliably recorded, and was only available for 72% of patients; of these nearly 40% belonged to social class I or II; in 10% both parents were unemployed. These figures reflect the population of our catchment area which includes a wealthy region of north London.

Source and nature of referrals

The majority of referrals were of in-patients on the paediatric ward (55%); in-patients from other wards made up 12.5%. Two-thirds of referrals were made by paediatricians; other referrals came from a wide range of hospital specialists, most notably haematologists, but also neurologists, general psychiatrists, surgeons and oncologists.

Of the 159 children, 34 had poisoned or injured themselves. Reasons for referral of the remaining 125 children were grouped into six categories (Table 1). Consistently, the bulk of referrals in this group were either requests for help in the management of physical illness or were of children with physical symptoms of non-organic aetiology (42.8–77%). Disorders of sleep, appetite, bowel and bladder function (categorised as habit disorders) and child-care problems, including child abuse, made up a further important proportion of referrals. The latter often reflected professionals' concerns about parents' ability to care adequately for their child, and in these cases the child did not necessarily have symptoms.

Diagnoses

Diagnoses were made according to the Multiaxial Classification Scheme for Psychiatric Disorders in Childhood and Adolescence (Rutter *et al*, 1983). The most common diagnosis on Axis 1 (clinical psychiatric syndrome) was of adjustment reaction (Table 2). 'Special symptoms or syndromes not classified elsewhere' includes some disorders with physical symptoms of non-organic origin such as anorexia, enuresis and encopresis. About 20% of children could not be assigned a psychiatric diagnosis, but it is important to note that in some of these cases one or both parents did have a psychiatric diagnosis. On Axis 5 (abnormal psychosocial situations) many patients fell into more than one category. The most frequent diagnosis was of 'anomalous family situation' (35%) which includes single parent and reconstituted families.

Table 2
Diagnoses according to Axis 1 (clinical psychiatric syndrome) of the Multiaxial Classification Scheme

Diagnosis (ICD-9 no.)		
Adjustment reaction (309)	40	(32%)
Special symptoms not classified elsewhere (307)	24	(19%)
Disturbances of emotion specific to childhood (313)	9	(7%)
Physical conditions arising from mental factors (306)	7	(6%)
Neurotic disorders (300)	5	(4%)
Disturbances of conduct (312)	2	(2%)
Other	10	(8%)
No psychiatric disorder	28	(22%)

Table 3
Referrals to child psychiatry during period of study

	Year 1 (1984/85)	Year 2 (1985/86)	Year 3 (1986/87)	z, P[1]
Total	116	104	124	
Liaison	46	43	70	$z=2.59$, $P<0.005$
Self-poisoning/ injury	7	14	13	NS

[1] Comparison of referrals between the three years.

Family functioning was recorded as adaptive in only 20% of the families of children referred. The remainder were described using a variety of family therapy terms; examples are included in the Appendix.

Confirmation of the reliability and validity of both diagnoses and descriptions of family functioning was not possible owing to the retrospective nature of the study.

Intervention

The main therapeutic intervention used was family therapy. Other interventions included individual psychotherapy, behaviour therapy and parental counselling. Psychiatrists were involved in assessment and treatment more often than non-medical members of the child psychiatry team.

Referral patterns

The number of liaison referrals to the department of child psychiatry increased significantly over the three years of the study, while both the total number of referrals to the department and the number of referrals of self-poisoning or injury remained constant (Table 3). Table 3 includes the results of the statistical tests comparing referrals between the three years. The result for the proportion of liaison referrals shows a significant trend from Year 1 to Year 3. The odds of a new referral being a liaison one rather than a non-liaison one in the third year of the study were almost twice (1.9 times) that in the first year. The number of liaison referrals exceeded the number of non-liaison referrals during the last year of the study.

Discussion

A deliberate decision to improve child psychiatry liaison in a district general hospital appears to have led to an increase in liaison referrals. This increase was not attributable to an increased total referral rate or increased referral rate of patients who had harmed themselves.

As part of the restructuring of the child psychiatry department in 1984, a number of changes were made to the service which probably contributed to these results. Regular psychosocial ward rounds, improved and increased undergraduate and postgraduate teaching, regular policy meetings and an active research programme into psychosocial aspects of physical illness were introduced. As in Brown & Cooper's (1987) study of the reorganisation of an adult liaison psychiatry service, institutional changes may have led to improved interprofessional relationships, an increased awareness of the importance of psychological factors in physical illness and a more realistic and well defined expectation of the service provided.

All members of the multidisciplinary child psychiatry team have made a valuable contribution to the liaison service. However, child psychiatrists were involved more often than any other discipline in the management of liaison referrals. The majority of these referrals were related to the management of physical illness or to a physical complaint that might have been non-organic. The most common diagnosis was of adjustment reaction to major stress. Child psychiatrists, who have a background of medical training and skills in psychodynamic and systemic treatment interventions, are well placed to work with these children and their families.

Fortunately, it was possible to expand this liaison service without jeopardising the child psychiatric service to the community. In other areas, any decision to reorganise the service may be more difficult to make owing to increasing demands on scarce resources. However, Offord (1987) identifies psychological morbidity related to chronic illness in children as a key area for preventative work. The expertise of child psychiatrists may therefore be necessary if the well recognised and well described long-term sequelae of both admission to hospital and physical illness in children are to be prevented.

Assessment of the efficacy of psychiatric interventions in both organic and non-organic somatic conditions in children is an important area for further research.

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Appendix

The following case vignettes are presented as examples of the work of the liaison service.

Case 1

A 13-year-old girl was referred by the paediatrician because no physical cause could be found for her abdominal pain.

At assessment, the psychiatrist noted her to be an obese, rather passive adolescent. The history revealed recent school refusal; the mother had had a stillbirth 18 months previously.

The psychiatrist described the mother–child relationship as "over-involved"; the mother who appeared quite depressed frequently acted in an "over-protective" and "infantilising" way towards her daughter during both assessment sessions. Following assessment, the family were referred to their local child guidance clinic for family therapy.

Case 2

A 10-year-old girl was referred by the paediatrician following her admission with nephrotic syndrome. The child was terrified of needles, and presented extreme management problems when blood tests were required. These were compounded by her positive hepatitis B status, which understandably meant that staff were reluctant to put themselves at risk. She lived with a large extended family, and her relationship with her mother did not seem good enough to sustain her during her difficult time in hospital.

A behaviour programme was instituted by the psychologist to treat the needle phobia; two family therapy sessions were carried out by the psychiatrist; and in addition, staff were helped to deal with their anxieties in the psychosocial round.

Case 3

A boy aged two and a half years had a history of diarrhoea and vomiting. His mother, a young single parent who suffered from schizophrenia, had brought him to hospital, but had been unable to actively seek help. After sitting in the hospital hallway for some time, she was noticed by staff and the child was admitted. He was noted to be developmentally delayed and inappropriately showed no stranger anxiety. The family were referred to the child psychiatrist as staff were concerned about the mother's ability to care for the child. Assessment revealed that the mother's mental illness had resulted in physical neglect and emotional abuse of the child. Pica had led to the gastrointestinal upset.

Following treatment of the mother's acute psychosis by the general psychiatrist, mother and child were transferred to a family treatment unit where work was carried out to improve the mother–child relationship and support the mother in the practical care of her child. The family continue to be seen for regular follow-up by the child psychiatrist. Social services have been involved throughout.

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The Motor Disorders of Mental Handicap
An Overlap with the Motor Disorders of Severe Psychiatric Illness

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Among 236 in-patients in one hospital for the mentally handicapped, there was a significant relationship between the amount of motor disorder (rated using a comprehensive check-list) and the severity of mental handicap, the presence of associated psychiatric disorder and the use of neuroleptic medication. The population was fairly evenly divided between those currently, previously and never having received neuroleptic medication. All categories of motor disorder, including abnormal movements, were present in all three subgroups. Neuroleptic medication appeared to modify the expression of motor disorder rather than producing it *de novo*. The range and frequency of motor disorders was comparable with that in patients with severe psychiatric illness. A common cerebral basis for the motor disorders of patients with mental handicap and severe psychiatric illness is suggested.

Motor disorder is a significant feature of people with severe mental handicap and is similar to the motor disorder of patients with severe psychiatric illness, but literature on this topic is limited. Early work, reviewed by Earl (1934), recognised this overlap. Plaskuda in 1911, for example, felt that the motor disorders of patients with severe mental handicap could be regarded as catatonic, but not as evidence of dementia praecox, since otherwise 60% of these patients would have been given this diagnosis. Steck (1926, 1927) included the motor disorder of mental handicap in his wide-ranging study of the extrapyramidal disorders of mental illness in which he tried to establish their cerebral basis. Earl (1934) studied 135 male patients with severe mental handicap and found motor abnormalities in practically all. He described various signs of catatonia, including catalepsy, echolalia, echopraxia, disorder of tone, flexion postures such as bowing of the head or the 'intrauterine' position, mutism, negativism and repetitive, rhythmical and semi-purposeful movements, as well as sudden outbursts of violent impulsive activity. He considered this symptom complex as identical to that found in patients with schizophrenia and argued against the psychoanalytic interpretations of motor disorder, which were being suggested at that time in both mental handicap and schizophrenia.

A psychological approach, however, became predominant. O'Gorman (1979), for example, favoured a psychogenic explanation for the vast majority of motor abnormalities present in mental handicap, although he invoked organic factors to explain the increased liability to motor disorder of the more severely retarded. With this psychological approach went a lack of detailed description of individual motor disorders. Ricks (1980) simply divided motor disorders associated with mental handicap into two forms: retarded, with apathy and little spontaneous movement; and cerebral palsy, with spasticity, ataxia or athetosis. Abnormal movements in the mentally handicapped were often grouped together as 'self-stimulation', with no study of their prevalence (Kalachnik, 1984).

Abnormal movements in the mentally handicapped attributed to neuroleptic medication, on the other hand, were the subject of several studies. None of these, however, ever included untreated individuals as a control group, so the prevalence of similar disorder in the drug-free remained unknown. Richardson *et al* (1986), for example, studied the 299 of 721 residents in an institution for the retarded who had received neuroleptics for at least one year in the previous five, and found a point prevalence of tardive dyskinesia of 30%. They compared this with the similar prevalence of 'tardive dyskinesia' which had been found in apparently untreated schizophrenics (Owens *et al*, 1982). They felt this similar prevalence cast doubt on the possibility of dyskinesia as part of the disease process of schizophrenia, rather than questioning their assumption that dyskinesia in treated mental handicap patients was entirely due to medication.

The position is changing. Youssef & Waddington (1988) found orofacial dyskinesia in 11 of 42 adult patients (26%) with mental handicap treated with neuroleptics, but also in two out of seven patients (29%) who had never received any. There was a significant association between orofacial dyskinesia and degree of mental handicap, which they took as reflecting greater cerebral dysfunction. They suggested that neuroleptics may enhance, or cause to arise prematurely, a motor syndrome that could emerge spontaneously both in mental handicap and patients with schizophrenia. A similar suggestion for patients with schizophrenia had been made by Steck (1954) in the first paper to describe Parkinsonism in such patients after treatment with neuroleptic medication, following his previous extensive study of extrapyramidal disorder in untreated schizophrenic patients. In this previous study (1926, 1927), he had suggested that the extrapyramidal disorder of both mental handicap and schizophrenic illnesses was due to dysfunction of the basal ganglia and frontosubcortical connections.

The present study is a comprehensive survey of the prevalence of different motor disorders in residents in an institution for the mentally handicapped. It is modelled on, and compared with, a previous study of patients with severe psychiatric illness carried out by one of the investigators (Rogers, 1985). As in the previous study, no attempt is made to separate 'neurological' and 'psychiatric' motor disorder *a priori*, but rather a straightforward description of the motor disorders found is given. Once both medication-based and disease-based motor disorder are regarded as expressions of cerebral disorder, dividing motor disorder, *a priori*, into 'neurological' and 'psychiatric' or into drug-induced and disease-induced as exclusive categories is arbitrary. The descriptive terms normally used make this arbitrary division almost inevitable and so are avoided as much as possible.

In both studies, a criterion of severity was adopted by choosing patients with prolonged continuous admission in one hospital, since it was felt that such patients would show the most severe motor disorder. Again the influence of various factors, including treatment and admission, on the motor disorder is examined.

Method

All 244 in-patients of one hospital for the mentally handicapped on a given day were chosen for study. The examinations were carried out over six months. Eight residents were excluded as they had been discharged.

This study was modelled on the previous study of motor disorder in patients with severe psychiatric illness (Rogers, 1985). Nursing staff on the wards were given a specific set of questions and each patient was observed, interviewed and examined by two of the investigators. Only features agreed as definitely present by both were counted as positive. A check-list of some 70 disorders under 10 different categories of motor disorder was used:

(a) *Posture:* head flexion, upright and lying; marked dorsal kyphosis; arm and finger flexion; hyperpronation/abduction/hyperextension of arms or trunk; persistence of posture
(b) *Tone:* increased or decreased in neck and arms; spasticity; *gegenhalten*; cogwheeling
(c) *Purposive movement:* general underactivity; help with washing, dressing or eating; slowness/freezing/abruptness of movement; paresis; difficulty with whole body/arm/finger movements on request; echopraxia
(d) *Activity:* general overactivity; outbursts shouting/threatening/hitting; objectionable/destructive/delinquent; self-injury
(e) *Movements of head, trunk or limbs:* brief random; semipurposeful; rhythmic; tremor; complex/simple tics; myoclonic; sustained spasmodic
(f) *Gait:* slow; shuffling; no/exaggerated associated movements; spastic; ataxic; walking aid needed; unable to walk
(g) *Eye movements:* roving; abrupt conjugate; to and fro; no eye contact; staring
(h) *Blinking:* increased; decreased; rapid bursts
(i) *Orofacial movements:* spasmodic contractions; fluid random; lack of expression; rhythmic/tremor/tics
(j) *Speech production:* mutism; partial mutism; unintelligible; dysarthria; volume/timbre/rate; stuttering/palilalia; echolalia

Posture was examined with the patient upright, and then lying in bed. Tone (resistance to passive movement) was examined in the neck and upper limbs: if increased it was graded as severe (complete rigidity). Increase in tone proportional to the strength of passive movement was designated '*gegenhalten*'. Purposive movement was examined by questioning the nursing staff; by direct observation; by examining for echopraxia (imitation) of arm and finger movement; and by asking for whole body, arm and finger movements. Abnormal activity was examined by questioning the nursing staff and by direct observation. Abnormal movements, gait and blinking were examined by direct observation. Speech production was examined by questioning the nursing staff and at interview. In addition a formal neurological examination was performed on each patient.

Independently, CB reviewed the patients' case notes and treatment charts. Information obtained included severity of mental handicap (as defined by ICD: mild, IQ 50–70; moderate, IQ 35–49; severe, IQ 20–34; and profound, $IQ<20$), associated psychiatric disorder, presence of epilepsy, and use of neuroleptic or other medication.

For statistical analysis of the results, Student's *t*-test, the χ^2 test, Fisher's exact probability test, and Pearson's product–moment correlation were used as appropriate. Only P values of less than 0.01 were considered significant.

Results

There were 204 men and 32 women, with an age range of 24–82 years (mean 50.1 years). The mean length of admission was 36.6 years (range 1–77 years), and the mean age at admission was 13.5 years (range 1–69 years). Fifteen per cent had mild retardation, 22% moderate, 23% severe, and 40% profound retardation.

Table 1
Superimposed psychiatric disorder, and treatment with neuroleptic medication

	No.	(% of whole group)
Any psychiatric disorder	104	(44%)
Behaviour disorder	73	(31%)
Affective disorder	23	(10%)
Psychotic disorder	19	(8%)
Other (e.g. anxiety, obsessive-compulsive disorder)	9	(4%)
Current treatment with neuroleptics	76	(32%)
Previous but not current treatment with neuroleptics	61	(26%)
No record of treatment with neuroleptics	99	(42%)

Salient features of the medical history included significant visual problems in 13%, significant hearing impairment in 15%, and a prevalence of epilepsy of 27%. As expected, superimposed psychiatric disorder was common (Table 1). The population was fairly evenly divided between those currently, previously and never having received neuroleptic medication, and 60% were taking other medication, including anticonvulsants in 25% and other neurotropic medication in 19%.

The study was designed to determine the frequency of motor disorder in a hospital population for the mentally handicapped and compare the results with those obtained from 100 patients hospitalised with severe psychiatric illness (Rogers, 1985): a similar range and frequency of abnormality were found (Table 2).

There was an abnormality of posture in 70%: 48% had flexion of the head when they were lying on their bed with no pillow, 19% had head flexion when upright, 13% had a marked dorsal kyphosis, 24% had arm flexion, 12% had finger flexion, 14% had hyperpronation/abduction/hyperextension postures of the arms or trunk, and 4% had persistence of postures after active or passive movement.

Table 2
Percentage of patients with abnormalities in each of 10 categories of motor disorder

	Present study (236 patients with mental handicap)	Rogers (1986) (100 patients with severe psychiatric illness)
Abnormality of:		
posture	70%	86%
tone	52%	85%
purposive movements	98%	97%
activity	62%	64%
movements of head, trunk or limbs	44%	67%
gait	68%	48%
eye movements	38%	48%
blinking	18%	38%
orofacial movements	63%	74%
speech production	89%	95%
Abnormality in:		
at least 1 category	100%	100%
at least 5 categories	73%	91%

A disorder of tone was present in 52%: 26% had increased tone in the neck (in 15% this was severe), 25% had increased tone in the arms (in 13% this was severe), 11% had decreased tone in the neck, and 15% decreased tone in the arms. Nine per cent showed spasticity, 6% *gegenhalten* and 1% cogwheeling.

A disorder of purposive movement was present in 98%: 18% were generally underactive, 54% needed help or encouragement with washing and dressing and 16% with eating, 23% had slowness, freezing or abruptness of movement, and 10% had paresis of one or more limbs. On request, 88% had difficulty with independent finger movements, 41% difficulty with arm movements and 8% difficulty with whole body movements. Forty per cent showed echopraxia. Of the 36 patients with significant hearing impairment, 92% had difficulty with finger movements on request, 39% with arm movements and 19% with whole body movements.

There was abnormal activity in 62%: 15% had general overactivity, and 43% had outbursts of abnormal activity with shouting and swearing, 27% with threatening behaviour, and 23% with actual assaults on others. Eighteen per cent had objectionable habits such as smearing faeces, 16% were destructive, 14% 'delinquent' and 15% had a history of self-injury.

Abnormal movements of the head, trunk or limbs occurred in 44%: in 7% these were brief and random, in 24% semipurposeful and in 11% rhythmic; 4% had a tremor, 10% showed complex tics and 2% simple tics. One patient had myoclonic jerking, and 4% had sustained, spasmodic movements.

There was an abnormality of gait in 68%: in 24% the gait was slow and in 28% shuffling. In 38% there were no associated movements of the arms and in 3% exaggerated associated movements. Six per cent had a spastic gait and 1% an ataxic gait. Five per cent needed a walking aid, 3% could only crawl and 5% could only use a wheelchair.

Abnormal spontaneous eye movements were present in 38%: 25% had roving, semipurposeful movements, 13% had abrupt conjugate deviation of the eyes, 2% had 'to-and-fro' movements. In 9% there was no eye contact with the examiner and 3% stared. Of the 31 patients with significant visual impairment, 52% had abnormal spontaneous eye movements, 26% had roving, 19% abrupt conjugate and 13% 'to-and-fro' movements; none avoided eye contact and 3% stared.

An obvious abnormality of blinking was present in 18%, with increased blinking in 9%, decreased in 6% and rapid bursts of blinking in 4%.

There were abnormal orofacial movements or an abnormal facial posture in 63%: 27% had spasmodic contractions of one or more facial muscles, 28% had fluid random movements, mainly round the mouth, 17% had a lack of facial expression, and 8% had rhythmic movements, tremor or tics of the orofacial muscles.

An abnormality of speech production was present in 89%: 8% were totally mute, 42% were partially mute, 46% had unintelligible speech, 36% had dysarthria, 15% had impaired volumes, timbre or rate of intelligible speech, 4% had stuttering or palilalia, and 6% had echolalia. Of the 36 patients with significant hearing impairment, 92% had an abnormality of speech production, 14% were totally mute and 42% partially mute, 42% had unintelligible speech, 47% dysarthria, 11% impaired volume, timbre or rate, 3% stuttering or palilalia and 9% echolalia.

On neurological examination, apart from motor disorder, there was an abnormality in 55%, with abnormality of tendon reflexes in 44%, extensor plantar responses in 15% and pupillary abnormality, squint or nystagmus in 19%.

In this group of 236 patients with mental handicap, therefore, there was a significant amount of motor disorder. All patients had an abnormality in at least one of the ten categories of motor disorder, and 73% had abnormalities in at least five categories.

To consider the association of motor disorder with other characteristics of these patients, each patient was given a motor score out of ten, according to the number of catgories with abnormality. There was no significant correlation between this motor score and age or length of admission, and no significant association with abnormality on conventional neurological examination or the use of non-neuroleptic medication.

There was a significant positive correlation between the motor score and the severity of mental handicap ($r=0.55$, $P<0.001$) and a significantly higher motor score in those with epilepsy ($t=3.64$, $P<0.001$), in those with a case-note diagnosis of associated psychiatric disorder ($t=3.67$, $P<0.001$) and in those who had ever been treated with neuroleptic medication ($t=4.56$, $P<0.0001$).

There was a significant positive association of degree of mental handicap and behaviour disorder, the largest component of associated psychiatric disorder ($\chi^2=28.11$, $P<0.001$), but not with any other psychiatric disorder. There was a significant positive association of current or past use of neuroleptic medication with both degree of mental handicap ($\chi^2=19.03$, $P<0.001$) and associated psychiatric disorder ($\chi^2=53.77$, $P<0.001$).

Treatment with neuroleptic medication at any time was significantly associated with the following individual motor abnormalities: hitting outbursts ($\chi^2=21.17$, $P<0.001$), destructiveness ($\chi^2=18.36$, $P<0.001$), threatening outbursts ($\chi^2=16.88$, $P<0.001$), general overactivity ($\chi^2=15.72$, $P<0.001$), verbal outbursts ($\chi^2=14.67$, $P<0.001$), no arm movement on request ($\chi^2=14.31$, $P<0.001$), help needed for washing and dressing ($\chi^2=14.27$, $P<0.001$), self-injury ($\chi^2=12.91$, $P<0.001$), objectionable habits ($\chi^2=11.00$, $P<0.001$), lack of eye contact ($\chi^2=10.76$, $P<0.01$), unintelligible speech ($\chi^2=8.96$, $P<0.01$) and spasmodic facial movements ($\chi^2=8.54$, $P<0.01$). There was no association between treatment with neuroleptic medication at any time and any of the remaining individual motor disorders examined.

Table 3
Percentage of patients in each of the three treatment groups – currently receiving, never having received and only having previously received neuroleptic medication – with abnormalities in the 10 categories of motor disorder

Abnormality	Current ($n=76$)	Never ($n=99$)	Past ($n=61$)
Posture	74%	62%	80%
Tone	55%	54%	46%
Purposive movement	97%	97%	100%
Activity	83%**	39%**	72%
Movements of head, trunk or limbs	53%*	32%	52%
brief random	12%	3%	8%
semipurposeful	24%	19%	33%
rhythmic	16%	6%	15%
tremor	5%	5%	0%
complex tics	9%	4%*	18%
sustained spasmodic	4%	3%	7%
Gait	63%	71%	69%
Eye movements	46%*	25%*	48%
roving	28%	18%	36%
abrupt conjugate	18%	8%	15%
blinking	21%	12%	23%
Orofacial movements	71%*	52%	70%
spasmodic contractions	32%	17%*	38%
fluid random	34%	27%	20%
rhythmic, tremor, tics	11%	5%	8%
Speech production	89%	85%	93%

Significant differences between pairwise comparison in adjacent columns with χ^2 test (no significant differences between current and past use of neuroleptic medication for any motor category) $^{*}P<0.01$, $^{**}P<0.001$.

The relationship with neuroleptic medication was further analysed by comparing the motor disorder in patients currently receiving, only previously having received, and never having received neuroleptic medication. The percentage of patients in each of these three treatment groups with abnormalities in each of the ten categories of motor disorder is given in Table 3. This shows that disorder in every category was present in each of these three treatment subgroups, including the 99 patients who had apparently never received any neuroleptic medication. There was a trend in most categories for abnormalities to be more common in those currently or previously treated, and in the categories of abnormal activity, abnormal movements of head, trunk and limbs, abnormal orofacial movements and abnormal eye movements, this difference achieved statistical significance. The percentage of patients in each of the three treatment subgroups with different abnormal movements of head, trunk, limbs, face or eyes is given in Table 3. This again showed a trend fot abnormal movements to be more common in those currently or previously treated with neuroleptic medication but this only achieved statistical significance for complex tics involving head, trunk or limbs and spasmodic movements of the face

Discussion

This clinical study demonstrated a significant amount of motor disorder in the residents of one hospital for the mentally handicapped, chosen for the severity of their mental handicap. Neither age-related cerebral changes nor length of institutionalisation were significant contributing factors to their motor disorder. There was no significant association between this motor disorder and abnormality on conventional neurological examination or use of non-neuroleptic medication, nor any significant contribution from impairment of vision or hearing.

There was, however, a significant association between the amount of motor disorder and the severity of the mental handicap, the presence of epilepsy, a case-note diagnosis of associated psychiatric disorder, and the use of neuroleptic medication at any time.

If the severity of mental handicap is a reflection of underlying cerebral dysfunction, then a relationship is suggested between motor and cerebral disorder. This is supported by the association of motor disorder with epilepsy. The association of motor disorder and psychiatric disorder probably has several causes.

The significant association between the amount of motor disorder and the use of neuroleptic medication could reflect either a direct effect of this medication or the fact that patients with more severe mental handicap were more likely to have motor disorder and more likely to receive neuroleptic medication because of their associated psychiatric disorder. There was a definite trend for different motor disorders to be more common in those patients who had been treated with neuroleptic medication, suggesting that neuroleptic medication made the expression of these motor disorders more likely. All these disorders, however, were also found in patients who had never received such medication and there was no particular disorder which could be wholly attributed to neuroleptic medication.

There was evidence, in fact, that neuroleptic medication did not make a major contribution to the amount of motor disorder found. Firstly, disorder in each of the ten categories was present in a considerable proportion of patients never treated with neuroleptic medication. Secondly, when the association of use of neuroleptic medication with individual motor disorders is considered, more than half of the significant associations are with components of abnormal activity. These were hitting outbursts, destructiveness, threatening outbursts, general overactivity, verbal outbursts, self-injury and objectionable habits. General overactivity could represent drug-induced akathisia, but other components could well be the cause of neuroleptic medication being prescribed rather than being caused by the medication. Thirdly, the remaining individual motor disorders significantly associated with neuroleptic medication – no arm movement on request, help needed for washing and dressing, lack of eye contact, unintelligible speech and spasmodic facial movements – do not suggest a consistent or expected pattern of medication effect such as Parkinsonism or tardive dyskinesia.

Abnormal movements of head, trunk or limbs were more common in those currently or previously treated with neuroleptic medication, but when the association of individual abnormal movements with neuroleptic medication is considered, the most significant association is with complex tics, which, psychiatrically, might be called 'stereotypies'. Likewise, abnormal orofacial movements were more common in those currently or previously treated, but here the most significant association was with spasmodic movements, which psychiatrically might be described as 'grimacing'.

When fluid, random, mainly peri-oral movements of the face, which, neurologically, might be described as 'dyskinesia' are considered, these were present in 34% of those currently receiving neuroleptic medication, 20% of those who had previously received such medication and 27% of those who had never received any neuroleptic medication. This should be compared with the average 29% prevalence of tardive dyskinesia reported in four different studies from 1975 to 1982 in 225 mentally handicapped subjects, all treated with neuroleptic medication (Kalachnik, 1984) and the 30% prevalence in the 299 treated subjects in the study by Richardson *et al* (1986). No future study of tardive dyskinesia in the mentally handicapped should omit a control group of similar patients untreated with neuroleptic medication.

Thus neuroleptic medication is neither a necessary or sufficient cause of motor disorder in these patients, and presumably other patients, with severe mental handicap, as was suggested for patients with severe psychiatric illness in the previous study (Rogers, 1985). It is not suggested that neuroleptic medication does not cause motor disorder in these patients, but rather that in patients with severe pre-existing cerebral disorder, including extrapyramidal motor disorder, it is more sensible to regard the effect of the medication as enhancing or modifying pre-existing motor disorder.

The spectrum of motor disorder found in this study of mentally handicapped in-patients was comparable with that found in the previous study of 100 patients with severe psychiatric illness (in hospital for an average of 43 years, 92% with a diagnosis of schizophrenia). A similar range and frequency of motor disorders were found in both groups of patients. In general, the psychiatric group had a greater prevalence of the different disorders, but this was not invariable. Difficulty performing upper-limb movements, echopraxia, mutism or very reduced speech, outbursts of abnormal activity, complex tics and sustained, spasmodic movements of head, trunk or limbs were more common in the mental handicap group. Paresis of one or more limbs and dysarthria were only found in the mental handicap group.

All the patients in both groups had disorder in at least one of the ten categories of motor disorder assessed, and some three-quarters in both groups had abnormalities in at least five of the ten categories. In neither study could motor disorder be related causally to length of admission or use of neuroleptic medication, although it is likely that both these factors modify the expression of motor disorder. Psychological explanations of motor disorder are becoming increasingly less acceptable. The most satisfying way of explaining the motor disorders in patients with mental handicap and those with severe psychiatric illness is as an expression of their underlying cerebral disorder. The bulk of these motor disorders would best be classified as extrapyramidal disorders. The overlap between the motor disorders found in both studies suggests that the cerebral disorder of patients with severe mental handicap and those with severe psychiatric illness includes a significant extrapyramidal disorder.

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Assessing Schizophrenia in Adults with Mental Retardation

A Comparative Study

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Twenty-five patients with mild mental retardation were compared with 26 schizophrenic patients of normal intelligence. The patients with retardation had suffered an earlier onset and were less likely to have been married or employed. However, the clinical phenomena exhibited by the two groups, as elicited by a SADS–L interview, were very similar. Thus, standardised interviews and criteria for schizophrenia can be readily applied to those with an IQ between 50 and 70.

The detection and diagnosis of psychotic illnesses in people with mental retardation is often thought difficult. The emergence in early adulthood of bizarre speech and behaviour can wrongly be attributed to the mental retardation itself, leading to delayed or inappropriate treatment. A brief case history is illustrative.

> A 25-year-old woman had shown early developmental delay and had attended schools for children with learning difficulties. At age 20 she had an IQ of 56 and lived in a convent with other mentally handicapped women. She developed outbursts of temper and appeared moody, but would not say why; a behavioural programme was implemented, but over 18 months she became more withdrawn and suspicious. When finally referred to a psychiatric out-patient clinic, she complained of voices saying "nasty things" about her much of the time, and described people putting thoughts into her head. The diagnosis of paranoid schizophrenia was made, and treatment with oral pimozide led to an excellent remission.

In this case, the diagnosis was clear by the time of psychiatric referral, but the difficulties of assessing patients with mental handicap have led some psychiatrists to question whether the usual diagnostic criteria for schizophrenia can be reliably applied.

Turner (1989) demonstrated that surveys of mental illness among the mentally retarded have often been vague as to the diagnostic criteria used. Reference is made to the Research Diagnostic Criteria (Spitzer *et al*, 1975) or DSM–III (American Psychiatric Association, 1980) in some more recent work (e.g. Gostason, 1985). However, even where relatively precise diagnostic guidelines are stated, there are grounds for thinking that they are not applied in a straightforward way. For example, Hucker (1979) states: "We began with the assumption that the intellectual limitations of the mentally handicapped might preclude the expression of first-rank symptoms and consequently a wider definition of schizophrenia was used." Certainly, Schneider's first-rank symptoms seem elaborate constructions in conceptual and linguistic terms when considered in relation to the abilities of the mentally retarded. Consequently, attempts to elicit first-rank symptoms from an individual with a very low verbal IQ are likely to be futile. However, several surveys have included individuals with IQs of 50–70, among whom Tredgold (1970) states, although without supporting evidence, "the features of mental illness that do appear among the imbeciles of mild degree resemble those occurring in ordinary patients".

The epidemiological focus of much recent work has meant that details of the phenomenology of the psychotic individuals identified have not been reported. An exception to this is the work of Hucker *et al* (1979) which gives rates for the occurrence of certain types of hallucinations and delusions. However, these authors provided no comparison group of schizophrenics without mental retardation, and in the absence of such a group, one cannot judge if the coexistence of mental retardation and schizophrenia leads to a different clinical picture. The study reported here sets out to address this question by the systematic examination of the psychotic phenomena exhibited by patients with mild mental retardation and a clinical diagnosis of schizophrenia. This group is compared with an unselected group of patients with schizophrenia but without evidence of mental retardation.

Method

Patients with mental retardation and a clinical diagnosis of schizophrenia were identified from the caseloads of consultants working in the mental handicap services of several south London health districts. Clinical case notes and educational records were examined and the following inclusion criteria were applied: (a) age 18–65 years; (b) clinical diagnosis of schizophrenia (ICD-9 295.1–295.9); (c) history of mental retardation requiring attendance at special school; ~~and~~ (d) documented full-scale IQ between

50 and 70 on the Weschler Intelligence Scale for Child (WISC) or the Weschler Adult Intelligence Schedule (WAIS).

The comparison group comprised patients with schizophrenia without mental handicap who, like the first sample, were receiving long-term care. A case register was used to identify all persons living in Camberwell who, on a particular census day, were regular attenders (defined as attendance at least weekly for the last month) at a psychiatric day hospital or day centre. The patients selected were aged 18 to 65 years, had a clinical diagnosis of schizophrenia, and had not received special schooling for mental retardation. Patients were given the National Adult Reading Test (Nelson, 1983), a measure of pre-morbid IQ, to ensure their full-scale IQ was in the normal range. Consent was gained from both groups of patients and from parents or guardians where appropriate in those with mental retardation.

All subjects were interviewed using the life-time version of the Schedule for Affective Disorders and Schizophrenia (SADS-L; Endicott & Spitzer, 1978). Information was also obtained from the case notes. From this population were selected those who fulfilled the RDC criteria for schizophrenia. Symptom definitions used were as described in the SADS-L interview (Endicott & Spitzer, 1978). The patients with mental retardation were visited or seen at routine out-patient appointments by one of the team (TT); the controls were interviewed in day-care settings by another (SWL). Analysis was carried out using the SPSSx (SPSS Inc., 1983) and BMDP (Dixon *et al*, 1983) statistical computing packages.

Results

Of the 28 patients with mental retardation and a clinical diagnosis of schizophrenia, 25 fulfilled the RDC for schizophrenia. In selecting the comparison group, 50 Camberwell patients with a clinical diagnosis of schizophrenia were contacted, but 13 declined to participate. Of the remainder, 27 fulfilled the RDC criteria for schizophrenia; one was excluded on grounds of mental retardation, leaving 26 control patients.

The schizophrenics with mental retardation were younger than those from the comparison group (Table 1) and had an earlier mean age of onset of psychiatric illness. Both groups had an excess of males. Ten (38%) of the comparison group had been married at some time. Only one of those with retardation had been married and he was separated at the time of the study. The patients with retardation were unlikely to have been in employment in the previous five years. Twenty (80%) had been unemployed for five years or more; only three had worked for more than half of that period. Three of the comparison group had been employed throughout the previous five years and 8 (31%) had worked for at least half of that time. In 23 (92%) of the group with retardation and 24 (92%) of the comparison group, the course according to RDC was chronic or subchronic.

Table 1
Demographic features of the two groups

	Schizophrenics with mental retardation (*n* = 25)	Schizophrenics without mental retardation (*n* = 26)
Mean (s.d.) age: years	35.4 (13.9)	43.8 (12.7)*
Mean age (s.d.) at first episode of illness, years	22.5 (6.5)	26.8 (8.7)*
Ratio: male:female	16:9 (64%)	18:8 (69%)

*$P<0.05$, two-tailed *t*-test

The phenomena elicited by the SADS-L interview are presented in Fig. 1. It can be seen that there were considerable similarities between the two groups. For example, auditory hallucinations were universal in those without retardation and were the most frequently reported single symptom in the group with it. There was however a tendency for the patients with retardation to show less psychopathology (for 13 of the 16 symptoms). This was greatest for persecutory delusions (odds ratio 0.15, 95% confidence interval (CI) 0.03–0.78), and for formal thought disorder (odds ratio 0.29, 95% CI 0.08–1.01).

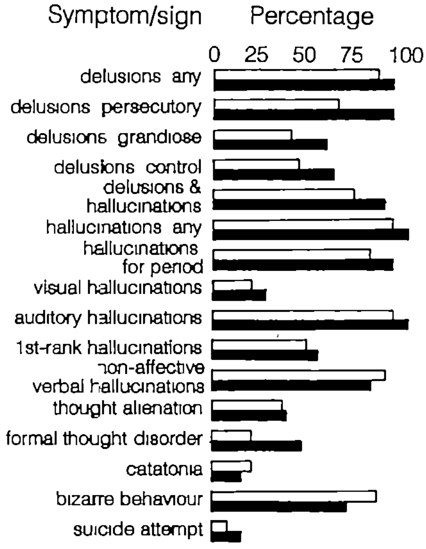

Fig. 1 Clinical phenomena (from SADS-L) in schizophrenic patients with (□) and without (■) mental retardation.

We attempted to establish whether the two groups could be distinguished on the basis of pattern of symptoms, by classical discriminant analysis (Hand, 1983*a*). The functions which this generated gave greatest weight to persecutory delusions and formal thought disorder (standardised canonical discriminant coefficients 0.56 and 0.55 respectively). Persecutory delusions showed the highest correlation with the discriminant function (0.44). The function correctly grouped 80% of cases. However, it is well known that the high rates found in initial design data sets are overoptimistic predictors of the performance of such functions in general

(Hand, 1983*b*). We therefore used an estimator of the true misclassification rate of the function (Efron, 1982). This showed that in general use the function would be expected to classify 61% of cases correctly. This is not very impressive since 50% would be expected to be correct by chance.

Discussion

To the best of our knowledge, this is the first time that a standardised diagnostic interview has been applied to a series of patients with both mental retardation and schizophrenia. Our experience was that the patients with mental retardation found the interview acceptable, and the results show that the phenomena elicited were similar quantitatively and qualitatively in schizophrenic subjects with and without mental retardation. This is encouraging both for researchers planning to use standardised diagnostic instruments in mildly mentally retarded populations, and for general psychiatrists unfamiliar with the assessment of schizophrenia in this group. Nonetheless, there was a suggestion that persecutory delusions, and perhaps formal thought disorder, may be less commonly encountered in patients with mental retardation. The latter finding may be a consequence of the reduction in ability to express things verbally which accompanies mental retardation.

The patients with mental retardation were less likely to have been married or to have been employed during the previous five years. Interestingly, the mean age at first episode of schizophrenia was significantly earlier in the group with mental retardation. This accords with findings of earlier onset of schizophrenia in subjects with a history of poorer academic achievement (Offord & Cross, 1972; Aylward *et al*, 1984). One explanation for this is that the existence of schizophrenia in the context of mental retardation is more likely to signal a neurodevelopmental disorder whose severity is reflected in the early appearance of psychotic symptoms. Alternatively, people with mental retardation may have a reduced functional capacity to deal with early symptoms of psychosis, hence presenting earlier.

In conclusion, it seems that standardised diagnostic instruments such as the SADS-L are acceptable and valid in the assessment of schizophrenia in mildly mentally retarded subjects. Moreover, the symptoms elicited are broadly similar in form and content to those found in schizophrenic patients of normal intelligence, although the timing and course of these symptoms may differ.

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Point of View

The Classification and Treatment of Dysthymia

D. G. M. MURPHY

The diagnostic concept and classification of chronic depressions has long been a matter of controversy. Kraepelin (1921) considered chronic depression to be a 'rudiment' of fully developed manic-depressive insanity, and this view was shared by Slater & Roth (1969), who cited occasional dramatic improvement in patients with neurasthenic disorder when they received convulsive therapy.

The opposing view, expressed by Schneider (1959), was that 'depressive psychopathy' has a constitutional aetiology, is on a spectrum with normal personality traits and types, and is not related to the affective disorders. This view was accepted by mainstream American psychiatry and in DSM–II (American Psychiatric Association, 1968) chronic depressions were considered to belong to the neuroses and personality disorders. In DSM–III however (American Psychiatric Association, 1980) the position was reversed and, in a theoretical shift, chronic depression was relabelled 'dysthymia' and moved from within the sections of personality and neuroses to that of the affective disorders.

Dysthymia is defined in DSM–III as follows:

(a) During the past two years (or one year for children and adolescents) the individual has been bothered most or all of the time by symptoms characteristic of the depressive syndrome but that are not of sufficient severity and duration to meet the criteria for a major depressive episode.
(b) The manifestations of the depression may be relatively persistent or separated by periods of normal mood lasting a few days to a few weeks, but no more than a few months at a time.
(c) During the depressive periods there is either prominent depressed mood or marked loss of interest or pleasure in all, or almost all, usual activities and pastimes.
(d) During the depressive periods at least three of the following symptoms are present:
    (1) insomnia or hypersomnia
    (2) low energy level or chronic tiredness
    (3) feelings of inadequacy, loss of self-esteem, or self-depreciation
    (4) decreased effectiveness or productivity at school, work, or home
    (5) decreased attention, concentration, or ability to think clearly
    (6) social withdrawal
    (7) loss of interest in or enjoyment of pleasurable activities
    (8) irritability of excessive anger (in children expressed towards parents or caretakers)
    (9) inability to respond with apparent pleasure to praise or rewards
    (10) less active or talkative than usual or feels slowed down or restless
    (11) pessimistic attitude toward the future, brooding about past events or feeling sorry for self
    (12) tearfulness or crying
    (13) recurrent thoughts of death or suicide.
(e) Absence of psychotic features, such as delusions, hallucinations, or incoherence, or loosening of associations.
(f) If the disturbance is superimposed on a pre-existing mental disorder, such as obsessive–compulsive disorder or alcohol dependence, the depressed mood, by virtue of its intensity or effect on functioning, can be clearly distinguished from the individual's usual mood.

The distinction between dysthymic disorder and major depression rests on the severity of depression, although the difference between the two syndromes as stated in DSM–III is just one symptom. In addition it failed to distinguish chronic depressions of early insidious onset from those secondary to other psychiatric or medical illnesses. The definition of dysthymia in DSM–III therefore lacked specificity. Rather than serving to define a clear relationship to the more 'biological' affective disorders, dysthymia – as defined in DSM–III – appeared to serve as a diagnostic catch-all.

An equivalent situation in general medicine may be postulated by inventing the term 'dyscardia', a condition which has bothered the individual for most of the past two years and whose symptoms are characteristic of the cardiovascular system but are not of sufficient severity to meet the criteria for a cardiac arrest:

(a) shortness of breath
(b) shortness of breath on exertion
(c) paroxysmal nocturnal dyspnoea

- (d) orthopnoea
- (e) central chest pain
- (f) palpitations
- (g) pain extending up into neck
- (h) Stokes Adam's attacks
- (i) vertigo
- (j) headache
- (k) transitory loss of consciousness
- (l) swollen ankles
- (m) progressive stepwise deterioration in cognitive ability.

From this it can be seen that a whole range of pathologically distinct disorders may be identified within the one group, and that the most basic of medical questions, such as "When did the pains start?" and "Have you had any illnesses or problems in the past?" are not addressed.

In an attempt to address this problem, further subclassifications of chronic depression have been proposed (Akiskal, 1983; Scott, 1988), and the diagnostic criteria for dysthymia have been modified in DSM-III-R (American Psychiatric Association, 1987). These schemes take account of the aetiological role of other physical and psychiatric illnesses, response to medication and age of onset.

In DSM-III-R the definition of dysthymia excludes those patients whose chronic depression is a residuum of a clearly defined major depression; it also provides two methods of subtyping dysthymia – primary versus secondary and early versus late. In addition, DSM-III-R allows dysthymia to be categorised with or without an accompanying major medical disorder:

- (a) Depressed mood (or can be irritable mood in children and adolescents) for most of the day, more days than not, as indicated by subjective account or observation by others, for at least two years (one year for children and adolescents).
- (b) Presence, while depressed, of at least two of the following:
  - (1) poor appetite or overeating
  - (2) insomnia or hypersomnia
  - (3) low energy or fatigue
  - (4) low self-esteem
  - (5) poor concentration or difficulty making decisions
  - (6) feelings of hopelessness.
- (c) During a two-year period (one year for children and adolescents) of the disturbance, never without the symptoms in (a) for more than two months at a time.
- (d) No clear evidence of a major depressive episode during the first two years (one year for children and adolescents) of the disturbance.

  Note. There may have been a previous major depressive episode, provided there was a full remission (no significant signs or symptoms for six months) before development of the dysthymia. In addition, after these two years (one year in children or adolescents) of dysthymia, there may be superimposed episodes of major depression, in which case both diagnoses are given.
- (e) Has never had a manic episode or an unequivocal hypomanic episode.
- (f) Not superimposed on a chronic psychotic disorder, such as schizophrenia or delusional disorder.
- (g) It cannot be established that an organic factor initiated and maintained the disturbance (e.g. prolonged administration of an antihypertensive medication).

Specify primary or secondary type:

> Primary type: the mood disturbance is not related to a pre-existing, chronic non-mood Axis I or Axis III disorder, e.g. anorexia nervosa, somatisation disorder, a psychoactive substance dependence disorder, an anxiety disorder, or rheumatoid arthritis.
>
> Secondary type: the mood disturbance is apparently related to a pre-existing, chronic, non-mood Axis I or Axis III disorder.

Specify early onset or late onset:

> early onset: onset of disturbance before age 21;
> late onset: onset of disturbance at age 21 or later.

The validity of this scheme is supported by recent work which demonstrates significant differences between patients with early-onset and late-onset dysthymia (Klein *et al*, 1988*a*,*b*). Subjects with early-onset dysthymia also appear to differ significantly from those with non-bipolar, non-chronic depression (Klein *et al*, 1988*b*).

Scott (1988) has suggested a four-part classification of chronic depression, in which dysthymic disorder covers a heterogeneous group of patients whose condition has an ill-defined onset in early adulthood and appears to be interwoven with the character style. In addition, Scott's classification system incorporates the work of Keller & Shapiro (1982) under the term 'double depression'. In this group acute major depressive episodes are superimposed on an underlying dysthymic disorder; the prognosis of the dysthymia is then poor and recurrence of major depressive episodes is frequent.

In the classification system proposed by Akiskal (1983), early-onset chronic depressive conditions are called 'characterological depressions'; these are further subdivided into two groups, those that are not responsive to medication (character-spectrum disorders) and those that are responsive to medication (subaffective dysthymic disorders). Akiskal argued that subaffective dysthymic disorder (SDD) represents a minor form of affective disorders whereas character-spectrum disorder (CSD) represents a form of personality disorder.

To support such a distinction, Akiskal *et al* (1980) have reported that patients with SDD have a short rapid-eye-movement (REM) latency, more continuous dysphoria, a positive family history for unipolar or bipolar depression, and depressive personality characteristics as described by Schneider (1958). Patients with CSD however were reported to be more commonly women, to have normal REM latencies, more intermittent dysphoria, positive family histories for alcoholism, a developmental history characterised by parental separation or divorce, and a dependent, histrionic or sociopathic personality (Akiskal *et al*, 1980, 1981; Rosenthal *et al*, 1981; Akiskal, 1983). These findings have important clinical and research implications; they therefore warrant re-examination.

Some of the literature published to support this proposed subclassification of dysthymia (SSD v. CSD) contains methodological shortcomings which may have some important effects on the measurement of medication responsiveness, REM latency, personality and family history.

Although the distinction between SDD and CSD is based upon responsiveness to medication, a standardised treatment regime was not used. In addition, there was no control or placebo group, no standardised rating scale for depression, no means of knowing if patients were taking medication, and none of the investigators was blind to treatment offered or outcome.

The finding of a decreased REM latency in patients with SDD is an important one and hints at a more 'biological' illness. However, there are a number of confounding variables which may have contributed to this result. For instance, it may be that at the time of assessment the group of patients with SDD were more depressed than those with CSD. This illustrates the desirability of using a standardised rating scale for depression. In addition details are not provided of the methodology employed during sleep electroencephalography (EEG) – particularly the drug-free period and number of adaption nights before measurement of REM latency.

It is proposed by Akiskal (1983) that those subjects with CSD have dependent, histrionic or sociopathic personalities whereas those with SDD have personalities which fit Schneiderian depressive typology. There are two possible objections to this proposition: firstly, in the context of a depressive condition, personality and its recall (by the subject) is a dependent variable; secondly, in those having a depressive illness of early onset, their 'personality disorder' may actually be a complication of longstanding depressive illness.

The family history of these subgroups is claimed to differ significantly, the CSD group having a family history of alcoholism and parental assortative mating while the SSD group have a family history of unipolar and bipolar illness (Akiskal, 1983). There are some potential sources of error which may have led to this proposition, among which are the reliability of the subjects as informants and the interpretation of results.

The subjects themselves may not be equally reliable informants for family history, as the experience of foster homes or orphanages was largely limited to patients diagnosed as CSD. In addition the CSD group had a significantly higher degree of developmental object loss than patients with SDD (Rosenthal *et al*, 1981).

Although the family history of 'total affective illness' (Akiskal *et al*, 1981) is significantly greater in the SDD group, there was no difference found in the proportions of family histories being positive for suicide. The conclusion drawn from this was that while less disposed to depressive illness, unstable adjustment in the kin of the CSD group – probably as a result of alcoholism – brought as much exposure to familial suicide.

Perhaps another interpretation is that there is no direct evidence that this was due to alcoholism *per se*, as there is no corroborating evidence quoted from autopsy reports or medical notes. This difference could possibly have been due to a combination of unrecognised depressive illness and the unreliable family history which may be obtained from a patient group with a high degree of developmental object loss. If most suicides in the relatives of those patients diagnosed as CSD are assumed to be due to affective disorder instead of alcoholism then there is no significant difference between groups with regard to family history of affective disorder. Against this latter proposition is the low frequency of bipolar illness in the relatives of patients with CSD (3.3%), as opposed to those with SDD (35%). However, only 2.5% of the unipolar control group used in this study had a family history of bipolar illness. It is a surprising finding that a subgroup of patients with dysthymia have a more 'biological' family history than the unipolar controls, and this result warrants further investigation with a larger sample.

Murphy & Checkley (1988*a*) attempted to confirm the validity of Akiskal's proposed classification system for early-onset CSD in an out-patient population of depressed patients. Significant differences were found between patients diagnosed as having a major affective episode and those having dysthymia with regard to anxiety, age at presentation and age at onset of illness, number of previous depressive episodes, family history and separation experiences before age 15. However, among the dysthymic patients, no significant differences could be demonstrated between those having subaffective dysthymic disorder or character spectrum disorder. The number of patients in this study is slightly smaller than in Akiskal's and further replication is needed.

People with dysthymia have experienced a long-term debilitating condition which saps the morale of both themselves and their therapist. Novel treatment approaches, both biological and psychological, are required. The new 5-HT_2 receptor antagonist ritanserin has been reported to significantly decrease the depressive symptoms of patients with dysthymia, in a double-blind placebo-controlled study (Murphy & Checkley, 1988*b*). A treatment effect was evident in both subgroups (CSD and SDD).

Conclusions

Dysthymia as defined in DSM-III is an ill-defined and non-specific diagnostic 'catch-all', with limited useful clinical application. Its main problem is that it lacks specificity. In order to address this problem, various subcategories of chronic depression have been proposed. Those systems that take account of age of onset and the role of other psychiatric or medical conditions seem to have some clinical and research applications. The DSM-III-R definition has received support from clinical studies. However, though the attempt to split early-onset chronic depressions into those that are primarily 'biological' (subaffective dysthymia) and those that are primarily 'characterological' (CSD) may make some intuitive sense, it is based on studies which have some methodological and theoretical drawbacks. At present the proposed subclassification (Akiskal, 1983) of the early-onset 'characterological' depressions has little clinical use; however, if this distinction were to be validated by further investigation it would be of considerable value insofar as it would identify patients liable to respond to drug treatment. It has been demonstrated that patients with dysthymia do respond to treatment with a novel antidepressant (ritanserin). Novel psychotherapeutic approaches are also required. A vigorous and optimistic treatment approach to this disorder is warranted.

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The Nithsdale Schizophrenia Surveys. VIII: Do Relatives Want Family Intervention – And Does It Help?

R. G. McCREADIE, K. PHILLIPS, J. A. HARVEY, G. WALDRON, M. STEWART and D. BAIRD

Sixty-three relatives of 52 schizophrenics living at home were offered a package of treatments by professionals working in an everyday NHS setting: educational seminars, relatives' groups, and family meetings. Thirty-two relatives refused intervention. Of the 31 relatives who agreed, 14 attended neither the educational seminars nor the relatives' groups. Seventeen relatives had a mean of ten treatment sessions, but there was little change in their level of expressed emotion after intervention. The number of patients who relapsed was the same in the 18 months before and after intervention, although the total number of relapses fell after intervention.

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A recent consistent finding in schizophrenia research is the impact family intervention has on relapse rates. Different forms of intervention in the UK (Leff *et al*, 1982, 1985, 1989; Tarrier *et al*, 1988, 1989), and in the US (Falloon *et al*, 1982, 1985; Hogarty *et al*, 1986), have produced a reduction in relapse rates which may be associated with a reduction in the level of relatives' expressed emotion (EE).

The reported studies have involved research teams in addition to clinicians responsible for patient care. "The application of the results in a routine way to the work of the busy clinical team must be the next phase" (Kuipers & Bebbington, 1988). The present study attempts to answer the following questions: in an everyday NHS setting, do relatives want intervention; does such intervention lower relatives' EE; and is there a reduction in relapse rates?

Method

The method of identifying schizophrenics has been described elsewhere (McCreadie, 1982; McCreadie & Robinson, 1987). Briefly, repeat censuses since 1981 in Nithsdale, an ancient and geographically discrete part of Dumfries and Galloway Region in south-west Scotland, have identified all known schizophrenics – in-patients, day patients, out-patients and patients known only to their family doctor. A repeat census on 1 January 1987 identified 77 patients living at home with relatives or friends: it was these on whom the present study focused. Patients were informed of the purpose of the study by the consultant responsible for their care (RGMcC) and if consent was given an approach was made by the consultant to the relatives with whom the patient was living. Frequently both these interviews were lengthy. If relatives then consented, a psychiatrist (KP) trained in the use of the Camberwell Family Interview rated EE in relatives and assessed the amount of face-to-face contact between patient and relative during a typical week. Relatives fell into the high-EE category if they made six or more critical comments, showed any degree of hostility, or had a rating of three or more on emotional overinvolvement (Leff *et al*, 1982); more than 35 hours a week indicated high contact (Vaughn & Leff, 1976).

Relatives were offered a package of treatments: educational seminars, relatives' discussion groups, and family meetings. The seminars, held weekly at the local psychiatric hospital, lasted approximately one hour, and were led by a psychiatrist of four years' experience (GW). They covered the main areas of aetiology, symptoms, treatment, and prognosis. Relatives were encouraged to ask questions. The relatives' groups, also held weekly at the hospital and lasting one to one and a half hours, were led by two social workers who had been qualified for seven and six years, and were both mental health officers (JAH and DB). One had attended a one-year course on family therapy on a day-release basis at the Scottish Institute of Human Relations, Edinburgh. The groups focused on the day-to-day problems of coping with a schizophrenic relative. One principal aim was to bring high-EE and low-EE relatives together, and encourage low-EE relatives to offer their solutions. Family meetings, which included the patient, were held weekly or fortnightly, and were led by a social worker in the patient's home (JAH or DB). They focused on everyday problems.

Relatives' EE and amount of contact with the patient were reassessed at the end of treatment. The interviewer was not told if the relative had in fact taken part in the intervention programme (see below), nor did he ask the relative; inevitably, however, the topic arose on occasion during interviews.

Patients were followed for 18 months after intervention. The number of relapses in those 18 months and in the 18 months before intervention was determined. As in a previous study (McCreadie & Phillips, 1988), relapse was defined in two ways. Firstly, if a patient had been readmitted to in-patient care, his/her case records were scrutinised and a decision made as to whether that admission was the result of an exacerbation of a chronic schizophrenic illness or a fresh episode. Secondly, as not all patients who relapse are necessarily readmitted to in-patient care, medication prescribed to the patients over the 18 months was determined through examination of medication charts and out-patient records. A relapse was deemed to have occurred where the dose of an antipsychotic drug was increased by at least 100%, where a patient on no antipsychotic medication was then prescribed such a drug, or where another antipsychotic drug was added to the pre-existing antipsychotic regime. Assessment of relapse was made by a psychiatrist (MS) blind to both the EE ratings and whether or not the patients' relatives had had the intervention programme.

Results

All 77 patients had a firm clinical diagnosis of schizophrenia, and 62% fulfilled the Feighner criteria for 'definite' or 'probable' schizophrenia (Feighner *et al*, 1972). There were 35 men, with a mean age of 43 years (range 21–68), a mean length of illness of 13 years (<1–42 years), and a mean time since last hospital admission of 5 years (<1-21 years); and 42 women, with a mean age of 44 years (range 18–78 years), a mean length of illness of 15 years (<1–50 years), and a mean time since last hospital admission of 6 years (<1–30 years).

Relatives and/or friends of 25 patients were not interviewed: 10 patients lived with other schizophrenics and/or in a group home, six patients had made no contact in any of the previous Nithsdale surveys, three patients did not attend interview when consent was sought to approach relatives, one patient refused an approach to relatives, two relatives failed to keep appointments, a family doctor refused an approach to either a patient or her relatives, and two patients were not seen for administrative reasons. Thus 63 relatives of 52 patients were interviewed, of whom 31 agreed to intervention and 32 did not. There were no differences between those who did and did not agree in sex distribution, relationship to patient, and numbers with low EE (Table 1); and in the following patient characteristics: sex, age and length of illness. In the refusal group, relatives with high EE and those who declined an assessment of their EE status numbered 16, compared with 15 with high EE in the group agreeing to intervention. The principal reasons for refusing intervention were: 'things are fine at the moment' ($n=12$); 'it's the patient who needs help, not me' ($n=6$); 'the patient doesn't want anyone else to know he has been ill' ($n=5$). Other reasons given by a single relative included 'I'm too busy', 'I've coped all these years', 'it's too late now'.

Fourteen relatives (of 13 patients) who agreed to intervention failed to appear at the educational seminars and relatives' groups; they only had one or two family meetings in their homes. The remaining 17 relatives (of 12 patients) were designated the intervention group. They had a mean of 10 treatment sessions (range 3–20), viz. educational seminars (mean 2, range 0–4), relatives' groups (mean 3, range 0–8), and family meetings (mean 5, range 0–14). At least one member of each family had a minimum of eight sessions (mean 12, range 8–20).

Sixteen of the 17 relatives had their level of EE and amount of contact with the patient measured before intervention. Five had high EE/high contact, four high EE/low contact, two low EE/high contact, and five low EE/low contact. Thirteen relatives were reassessed at the end of intervention. One had changed from high to low EE, and one from low to high EE; the remaining 11 (six high EE, five low EE) were unchanged. Two relatives in high contact were now low, two in low contact high.

Table 1
Characteristics of relatives

	Relatives who agreed to intervention ($n=31$)	Relatives who did not agree to intervention ($n=32$)
Sex		
male	15	13
female	16	19
Relationship to patient		
father	7	4
mother	10	13
husband	4	8
wife	3	5
child	5	1
other	2	1
Level of expressed emotion		
high	15	9
low	15	15
refused	–	7
not known	1	1

No significant differences between groups

Eight of the 12 patients whose relatives took part in the intervention programme had been admitted to hospital during the previous year, compared with four of the 27 patients whose relatives refused intervention ($\chi^2=8.19$, $P<0.01$). Nine of the 12 patients in the intervention programme relapsed a total of 17 times in the 18 months before intervention; six relapsed a total of eight times in the 18 months after intervention. Readmission to hospital defined 23 of the relapses, a 100% increase in medication defined two. The number of relapses in the seven patients from high EE homes fell from 13 to 4 (sign test, $P<0.06$).

General comments

The relatives who took part in the intervention programme were in the main pleased with the results. They were initially nervous about attending the group, and needed encouragement from the group leader. They appreciated the educational seminars most and believed at the end they knew more about the illness, treatments available, and prognosis. They were pleased that 'for a change' interest was being shown in relatives. They thought the relatives' groups were most useful if there was a crisis at home. They were relieved their experiences were not unique. Topics frequently discussed included violence, loneliness, child-like behaviour, withdrawal, refusal to take medication, and anxiety over suicide attempts. Both in the relatives' groups and in the family meetings means of overcoming these problems were examined. Practical help, for example in organising holidays and financial advice, was appreciated.

During the 18-month follow-up most relatives believed that things were easier at home. Also, a loose network among relatives continued after formal intervention stopped. The network was strengthened when many relatives and patients joined either the local recently established Mental Health Association or National Schizophrenia Fellowship, and thus education and support, organised by and for patients and relatives themselves, continued. Relatives who refused intervention did not join either organisation.

Discussion

The present study is "different" (Bebbington, 1988) in that it examined schizophrenic patients in remission living in the community. Other studies of EE and family intervention have examined patients admitted to hospital after a relapse. Caution must therefore be taken when results in this study are compared with others.

Perhaps the most important finding was the difficulty experienced in engaging relatives in treatment. Most relatives did not want to participate in the family intervention programme. Many reasons were given, but the statement 'things are fine at the moment' occurred repeatedly. It was our subjective impression that in many cases this statement was accompanied by a mixture of resignation, pride and resentment. It is not suprising therefore that most relatives who did eventually agree to intervention were living with a patient who had been readmitted to hospital the previous year. A number of relatives said they would join an intervention programme, but did not then attend the educational seminars or relatives' groups. Both these activities took place during the day at the hospital, and this might have mitigated against attendance. In the study by Leff *et al* (1989), five of 11 families failed to attend a relatives' group in central London. Leff *et al* now believe it is particularly important to offer home visits to families who are unable or refuse to attend the relatives' groups. It is clear therefore that vigorous outreach is necessary in order to enable relatives to accept an offer of help, particularly if they feel other offers have been unhelpful.

Although relatives who did participate in the intervention programme had a considerable number of sessions in one of the three treatment areas, there was virtually no change in their level of EE after intervention. There are three possible reasons for this. Firstly, the intervention may have been inadequate or did not continue long enough. Treatment was given by professionals who were experienced practitioners, but who did not have special skills in family intervention in schizophrenia. They were aware of the principles behind the intervention of Leff *et al*, but had received no training from that group. The intervention in the present study is therefore an approximation of what was achieved in other studies; however, this probably reflects clinical reality, as it is unlikely that many professionals working in hard-pressed NHS psychiatric facilities will be seconded for specialised training. A second possibility is that after family intervention the level of EE falls only gradually, and that if a reassessment of EE had been carried out at the end of the 18-month follow-up results might have been different. However, a previous study (Tarrier *et al*, 1988) found EE status had changed by four and a half months after intervention. Thirdly, it may be that the level of EE is robust, and is a stable dimension. Even in intensive intervention (Leff *et al*, 1982, 1985, 1989) six of 12 and 10 of 16 relatives did not move from high to low EE. As the years pass in Nithsdale a considerable number of relatives have had their level of EE rated on a number of occasions, separated by many months. This will be the subject of a separate inquiry, but there is tentative evidence that the level of EE changes little over the years. In this context it is again necessary to emphasise that relatives were assessed when patients were not in a state of relapse.

Relapse was used as the outcome measure firstly because it allows easy comparison with most other previous studies, and secondly because it is readily understandable to clinicians. It might have been possible to use broader outcome measures such as family burden, problem solving, and role performance of patients. However, these more sophisticated measurements are not routinely applied in an everyday NHS practice. Although intervention did not greatly alter the number of patients who had at least one relapse, there was a fall in the *total* number of relapses over the subsequent 18 months, a fall which just failed to reach statistical significance at the 5% level in patients living in high-EE homes (the sample size was small). Consideration of the total number of relapses is probably realistic and of importance to relatives. Other studies have shown that relapse rates can fall without a corresponding fall in high EE. In an American study (Hogarty *et al*, 1986) this happened when social therapy and family intervention were combined; in a UK study (Leff *et al*, 1989) it happened when face-to-face contact was reduced. If the finding in the present study is genuine, that there has been a fall in the total number of relapses, what factors in Nithsdale might be important? It is possible that an improved ability among families to solve problems has reduced both the patients' vulnerability and their environmental stress. In addition, the much greater interest in relatives shown by doctors and social workers already well known to schizophrenic patients might have increased the family's sense of worth and support. Finally, the network among relatives established during follow-up provided continuing mutual support. It is noteworthy that although relatives believed the educational seminars to be most helpful, a previous study (Tarrier *et al*, 1988) found education alone had no effect on relapse rates.

In conclusion, if professionals working in everyday NHS facilities wish to embark on family intervention, they should probably offer such help to relatives living with patients who have recently been readmitted to hospital.

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Replacing the Mental Hospital

Community Provision for a District's Chronically Psychiatrically Disabled in Domestic Environments?

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Eighteen long-stay patients from an old county asylum moved into hospital hostels. After 12–24 months, there were significant reductions in overall symptoms, as rated by the CPRS and the Krawiecka scale, as well as in deviant but not general behaviour, rated by the REHAB scale. Six later moved to live in supported accommodation. All relatives and patients who expressed an opinion to an independent assessor were satisfied with their present accommodation or wished to move to more independent living.

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As the run down and closure of the mental hospitals proceeds, the need to provide the chronically psychiatrically disabled in hospital and in the community with continuing support and asylum has become increasingly apparent. The vast majority continue to require support from hospital services and only a very small proportion live in their own home or their family's. Both Italian (Jones & Poletti, 1985) and American (Brown, 1985) experiences confirm the need for those with chronic psychiatric disabilities to be provided with suitable accommodation with adequate levels of support staffing. Curson *et al* (1988) have shown that almost half of their long-stay schizophrenic patients have active symptoms and a "sizeable proportion exhibited behaviour which could set them apart in a community setting." They conclude that "inpatient facilities, albeit at a local level, will be required to offer genuine asylum for those patients who are most severely ill." The alternative of slowing down or even stopping the closure programme leaves a sizeable proportion of patients in hospital who, when they have been asked, have expressed the wish to leave hospital (Abrahamson *et al*, 1989).

A range of accommodation options is described in standard handbooks of rehabilitation practice (Wing & Morris, 1981), but descriptions of such ranges of facilities in operation are relatively few, and diverse in the types of facilities they describe. This range has been supplemented recently by newer developments such as hospital hostels for new long-stay patients (e.g. Gibbons, 1986), which are increasingly seen as suitable environments for providing asylum and support for this group of patients. An extension of this concept is the 'haven community' for the severely disabled described by Wing & Furlong (1987) "within the context of a comprehensive psychiatric community service". Development of such provision is likely to become more widespread in the future and would seem to be desirable in providing care within patients' own communities. The characteristics of a service which has developed an integrated and comprehensive range of residential care for the chronically psychiatrically disabled in a district which does not contain a mental hospital is described.

Description of the service

The initial establishment of the mental health services in Bassetlaw has been described by Kingdon & Szulecka (1986). Policy decisions were made to accept all new and old long-stay patients from Saxondale Hospital (the 'county asylum', which closed in 1988) who originated from and wished to return to the district. Domestic as opposed to ward provision for continuing and rehabilitation care was developed.

Hospital hostels sited on the edge of the site of the district general hospital were opened in June 1988 and October 1987 with the transfer of 18 patients from Saxondale and one new long-stay patient from the acute ward of the district general hospital. These are staffed 24 hours a day by nurses. All patients are registered with general practitioners and are regularly seen as out-patients by psychiatric staff, except in emergencies or where patients are unfit to attend a clinic, when they are seen in the hostels. Regular multidisciplinary reviews of individual patients also take place. Token-economy or similar reward systems are not used. The rehabilitation system is based on social rewarding with structured objectives.

Other supported accommodation comprised the following. Local-authority residential homes for the elderly have provided accommodation for long-stay patients discharged from hospital, including those under 65 years old who are frail, as specialised social-services accommodation for this group is not provided within the district. An adult placement scheme is being developed. Private residential accommodation includes nursing homes for the elderly, with a home specifically for those with organic brain disease, an eight-place psychiatric rest home run by nursing staff previously employed in the district service, and supported lodgings, which accept clients with long-term disabilities and whose landlords/ladies are supported by the multidisciplinary team in so doing. Supervised flatlets (10 places) were established in two houses bought by Nottingham Community Housing Association and run by the Turning Point organisation which provides staff for daily supervision and night cover. Warden-assisted accommodation is made available to patients where appropriate. Support in these and patients' own homes is given by a general psychiatry community team who were 'generalists', with one community psychiatric nurse specialising in rehabilitation. An application for funding to the housing department for a scheme to provide single housing with a 'roving' warden specifically for those with psychiatric disabilities has been made by a local housing association.

Two group homes were in existence before the establishment of the service. Both were initially run by a voluntary organisation (MIND). One was handed over to the housing department of the local authority with the resident's agreement, and so ceased to be a group home.

Clustering together of a small number of day-centre attenders for the purpose of mutual support into one small block of flats has been arranged by the local council as flats have become vacant.

Financial costs of the development

No special funding was available for the development from the Department of Health (DoH), etc. Trent Region's Strategic Reserve provided finance for the hospital hostels, which were established in two houses, one of which had been staff accommodation. The cost of buying the second and conversion of the two was £163 000, with £34 000 for furnishings and equipment. Running costs are less than £12 500 per patient per annum. This figure has been lower than planned because of difficulties in recruiting paramedical staff. Equivalent costs of a patient in a mental hospital are at least £13 200 per annum. The nursing and

Table 1
Diagnoses of patients transferred from asylum to more domestic settings

Diagnosis[1]	Transfer group ($n=18$)	New hospital hostel patients ($n=9$)	Others in supported accommodation ($n=17$)
Schizophrenia	7	2	11
Manic-depressive psychosis	4	3	3
Depressive neurosis	3	1	2
Alcohol dependence	1	1	0
Organic syndromes	3	1	0
Personality disorder			
histrionic/asthenic	2	3	0
antisocial	2	1	1
obsessional	0	0	1
Conversion hysteria	1	0	0
Bulimia nervosa	0	0	1
Mental subnormality (borderline)	0	1	0

1 According to ICD-9 criteria (World Health Organization, 1978). Note that some patients qualified for more than one diagnosis.

Table 2
Symptom characteristics of 17 long-stay patients transferred into the district

	REHAB scores		CPRS scores		Krawiecka scores
	general	deviant	total	global	
Before transfer (mean)	72.35	2.06	14.65	1.76	10.12
After transfer (mean)	67.88	1.12	8.00	1.12	4.29
Patient ratings at end of study period					
Hospital hostel (mean)	79.95	1.68	12.79	1.37	5.74
Other supported accommodation (mean)	41.54	1.39	10.09	1.44	4.04

rest-home accommodation attracts the standard rates set by the Department of Social Security (DSS). The Turning Point flats were bought and converted from a grant of £175 000 from the housing corporation (of the Department of the Environment) and £5000 for furnishings. Running costs are provided by the DSS (rest-home rates) with top-up funding of £20 000 per annum from the health and local authorities. The group homes were donated out of local council housing stock and rent is paid by the DSS. Clustering of flats together required minimal administrative costs. Allocation of places in warden-controlled and Part-III local-authority accommodation was dependent on local government finance. No specialist social-work staff were employed for the project. Domiciliary services were used for four months for two residents who lived together and to support one resident who lives with his mother in warden-controlled accommodation.

Method

The study population comprised, firstly, all new and old long-stay patients who were moved from Saxondale Hospital to the hospital hostels in Bassetlaw. They were interviewed by DK, KS, KM or DT at least one year before, and between one and two years after, transfer to the district. Secondly, all those patients with chronic psychiatric disabilities aged 18–65, living in supported accommodation in the district, were interviewed by KM or DT. They were assessed using the Comprehensive Psychopathological Rating Scale (CPRS), which covers the total range of psychopathology and is sensitive to change (Asberg *et al*, 1978), and the Krawiecka scale (Krawiecka *et al*, 1979) which provides a measure of those symptoms most prominent in chronic psychiatric disorders. Evaluation of rehabilitation was undertaken using the REHAB schedules of Hall & Baker (1983) completed by trained nursing staff in the hostels and by care staff, with the assistance of KM and DT in other settings. The Patient Attitude Questionnaire, developed by the Team for the Assessment of Psychiatric Services at Friern Hospital (North East Thames Regional Health Authority, 1990) was used to assess the opinions of the patients on the changes, and modified for use with relatives who were in contact with them. It was administered independently by EL.

Results

Sixteen new and old long-stay patients resident in Saxondale Hospital were transferred into the district out of a total of 19 who had originated from it (two had not wished to return to their district of origin and one patient died of complications of diabetes before the move). Two patients transferred who were not from the district. The group was predominantly elderly (average age 66.7 years), unmarried, and with a variety of diagnoses (Table 1). They had marked disability and distress (Table 2). Use was made of acute psychiatric facilities after exacerbation of psychotic illnesses. All patients however returned there after amelioration of symptoms. At the end of the study period, nine remained in the hostel, of whom two had been accepted for local-authority (Part-III) accommodation but were awaiting places. One had died, six had moved to less intensively staffed provision. There were significant reductions in scores on the CPRS (global: $t=3.096$; d.f. 16; $0.01>P>0.001$; total: Wilcoxon's rank sum test, $P<0.01$) and the Krawiecka scale ($t=4.419$; d.f. 16; $P<0.001$). Overall REHAB scores showed little change but scores for deviant behaviour improved significantly ($t=3.88$; d.f. 16; $0.01>0.001$).

The assessor was able to obtain meaningful responses from 13 patients using the Patient Attitude Questionnaire: nine were pleased with their new accommodation, preferred it to hospital, and were happy to remain there; four were not particularly happy where they were and would have preferred to be in their own homes rather than live in their present accommodation indefinitely. None expressed a desire to return to hospital. Of these relatives who retained contact with patients (eight) and who had visited them and therefore felt able to comment (six), none expressed dissatisfaction with the change of accommodation.

At the end of the study period, there were significant differences in REHAB general scores ($t=3.93$; d.f. 40; $P<0.001$) between those remaining in the hospital hostels and those in other accommodation (14 men, 3 women, average age 40.3 years), but no such differences were found in Krawiecka or CPRS ratings.

A study of those admitted to forensic facilities, including the local prison, regional secure unit and special hospitals, coinciding with the period October 1984 to October 1986 (Kingdon & Bakewell, 1988), detected one patient with mental illness who was resident in secure accommodation. One person from the district with no history of psychiatric contact was admitted from the courts to a special hospital in 1987. No request for transfer of Bassetlaw patients from any of these institutions has been received to date. Since the completion of that study one transfer to the regional secure unit has taken place.

Discussion

Ward accommodation has not proved necessary for the care of chronically psychiatrically disabled patients, apart from brief support from an acute psychiatric ward to treat exacerbations of underlying psychiatric illnesses. In those patients transferred from the traditional mental hospital, deviant behaviour and symptoms improved in the hospital hostels, such that six patients moved to less intensively supported 'resting places' specially developed for them and those community patients requiring care, in the manner described by Wing & Furlong (1987). The only comparable description in the literature is of the Massachusetts service (Zanditon & Hellman, 1981), which provides for most, but not all, its chronically disabled patients in a range of domiciliary settings. They also have followed the progress of patients returning to their district of origin from Boston State Hospital over four years (Dickey *et al*, 1981) and found that all but two of their 27 patients preferred their current life to that at the state hospital. Our British study supports those findings.

The health authority is one of the smallest in England and is co-terminous with local-authority boundaries; this has undoubtedly enhanced planning and liaison, although recruitment of staff has at times proved difficult and the economies of scale of larger districts are not available. The demographic details of the district suggest that the population is unexceptionable. It may be that this type of provision would not be suitable for other districts with markedly higher levels of morbidity unless supplemented by intensive-care facilities. Some of the patients described however had displayed severe behavioural disturbance, including fire-setting and violence to person and property, and one had previously been resident in a special hospital.

The major advantage of domestic care is that the rehabilitation setting approximates far better to a normal home. Parry-Jones (1988) comments that, in the 19th century, "the emulation of a domestic family model was undertaken in all types of institution", though with few examples of success. The aim then also was to provide more comfortable and individual settings for patients who can move up and down the 'staircase' of care, eventually settling at a resting place which they can cope and be relatively contented (Wing & Furlong, 1987).

Care in the community should provide a wider choice of facilities and support for patients and their relatives. Although houses and nursing homes can become understaffed and neglected by senior staff, they are less likely to become so than easily identified, large and inevitably impersonal hospitals which are restricted in the range of 'resting places' that they can provide. The costs involved are not extortionate, but detailed planning and negotiation with the local community is essential.

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Bipolar Affective Disorder Following Head Injury

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A patient developed distinct episodes of major depressive illness, schizophreniform psychoses and mania as well as focal epilepsy following head injury. Head injury may be directly causative in the development of affective psychoses, in this case secondary bipolar (mixed) disorder.

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Head injuries resulting from road traffic accidents are a major cause of cerebral damage, particularly in young adults. One estimate suggests an annual attendance rate for head injuries of about 2000 per 100 000 referrals to casualty departments, with about 250 requiring admission to general surgical wards (Jennett & MacMillan, 1981). The most common presentation is of closed head injuries; in open head injuries the intradural contents communicate with the outside through skull and scalp wounds usually as a result of a sharp and circumscribed impact. In the closed head injury, the effects of external trauma are enhanced by the undulating internal surface of the cranium, damaged protective coverings of the brain, cutting action of blood vessels, and shearing and rotation of the brain, thus producing widespread neuronal injury and multiple areas of focal damage (Crockard, 1983).

Not surprisingly perhaps, a number of psychiatric conditions have been reported following closed head injury, with or without any demonstrable neurological sequelae. The relationship is, however, not straightforward and has fuelled several debates, particularly in the development of psychosis. Some (e.g. Davison & Bagley, 1969) favour a direct aetiological role, while others prefer a constitutional predisposition as the main contributing factor (e.g. Lishman, 1973). Depressive psychoses occur most frequently, although schizophrenia, and less commonly mania, are also reported (Achte *et al*, 1969).

An unusual case with no psychotic vulnerability is reported here.

Case report

The patient was involved in a car accident at the age of 27 years and sustained multiple fractures, including closed head injury involving fractures of the right skull bones. He was unconscious for over 12 hours, and had retrograde and anterograde amnesias upon recovery. Before the accident, he had worked as a busy salesman and was described by his wife as being a 'very able' man. He had no previous history of psychiatric illness, alcoholism or any illness suggestive of cerebral disorder, and no psychiatric illness or epilepsy are reported in the family.

Following the accident, he developed an amnestic syndrome, headaches, anxiety, tearfulness, and was unable to work. In fact, he never regained employment, and although according to his wife he remained fairly well between relapses while on medication, he lived a restricted life, in the extremely caring atmosphere of his family. His symptoms gradually worsened, and two years after the head injury he had his first attack of severe depression, with insomnia, hypochondriasis, and suicidal ideation, which responded to a course of electroconvulsive therapy (ECT). At age 31, he developed focal epilepsy, associated with Jacksonian fits spreading from the left hand to the left arm, and occasional generalised tonic–clonic seizures with incontinence, and unconsciousness lasting 10 minutes. The fits were controlled with phenobarbitone and phenytoin, which were discontinued 20 years later with no recurrence. Between the ages of 32 and 35, he had three further episodes of depressive illness associated with three suicidal attempts; on each occasion he improved with ECT and antidepressants.

At 40, he developed auditory hallucinations which subsided spontaneously; a year later he took a further overdose of drugs in response to commanding auditory hallucinations. He was convinced the voices were transmitted to him via a broadcasting device, and that thoughts were being removed from and inserted into his mind. He had thought disorder, persecutory delusions, and believed that his body had been programmed by a computer. In addition, his affect was depressed. His IQ (Wechsler Adult Intelligence Scale) was 99, and on the Wechsler Memory Scale and Williams Memory Tests, results were in line with the IQ estimate, delayed recall was well preserved, and there was impairment of the verbal learning of new material. Bender Gestalt drawings were accurately reproduced. On the Eysenck Personality Inventory, his neuroticism score was high, his extraversion score low, compatible with a 'dysthymic personality type'. Physical examination, routine blood tests, lumbar puncture and electroencephalography (EEG) were all normal. A course of chlorpromazine relieved his psychotic symptoms. At the age of 48, he presented with similar symptoms associated with a depressive state which responded to trifluoperazine and amitriptyline. At 57 years, he again developed delusions of being controlled by a computer and of thoughts being removed from his mind by beams emitted by the computer, persecutory delusions, and third-person auditory hallucinations. Although routine investigations and EEG were again normal, a computerised tomography (CT) brain scan showed a generalised atrophy, predominantly in the frontal region (Fig. 1).

Depot flupenthixol decanoate failed to control his symptoms but he responded to trifluoperazine which he continued until he was 58, when he presented with a manic psychosis. On admission, he was elated, sexually disinhibited, insomniac, and had loud, pressured speech. He believed he was exceptionally gifted, and described grandiose plans on space travel. He showed a copy of a poem he had sent to President Reagan about the space race, and thought his exploits would make his whole family rich. No schizophrenic first-rank symptoms were elicited. Flupenthixol decanoate (40 mg every three weeks) and chlorpromazine (100 mg per day) resolved his psychosis, allowing his discharge three weeks later. He was readmitted two weeks later in a hypomanic state. He was overfamiliar, elated, grandiose, had pressure of speech and, quite uncharacteristically, he had been drinking heavily. Lithium carbonate and thioridazine induced complete remission, with no recurrence of symptoms two years later.

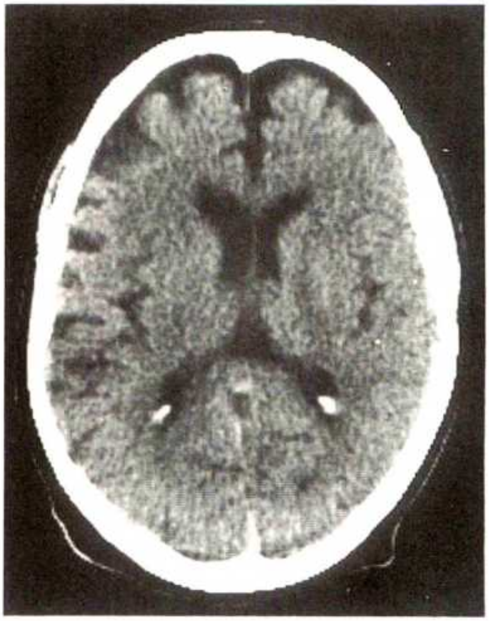

Fig. 1 Scan demonstrates evidence of generalised cerebral atrophy, with particular prominence of the cerebrospinal-fluid spaces over the frontal lobes. The ventricular system is normal in position.

Discussion

The case illustrates the heterogeneity of psychiatric symptoms resulting from brain injury in a patient who developed distinct episodes of major depressive illness, schizophreniform psychoses and mania. Although these syndromes may occur after head injury (Achte *et al*, 1969), there have been no reports of cases with such a variety of psychotic disorders occurring sequentially in the same patient following head injury. The association of schizophrenic symptoms with focal epilepsy is well documented. However, affective psychosis preceded the onset of epilepsy in this patient and seems to have resulted directly from the head injury and ensuing brain insult.

In a review of manic states occurring secondary to organic brain syndromes, Krauthammer & Klerman (1978) did not consider head injury as a causative factor despite previous reports supporting such an association. They have described four factors important in distinguishing secondary from primary mania: close temporal relationship between physical and mental illnesses, later age at onset of mania, absent pre-morbid characteristics, and lack of family history. Since their publication, a number of cases have been described which fulfil their criteria for secondary mania (e.g. Clark & Davison, 1987; Bracken, 1987; Shukla *et al*, 1987; Yatham *et al*, 1988). This patient developed a secondary bipolar (mixed) disorder with recurrent episodes of major depression and mania. We suggest that the case adds to the growing evidence that head injury may be directly causative in affective psychoses.

In keeping with the observation by Cohn *et al* (1977), we observed a good response to lithium at two-year follow-up. Clark & Davison (1987) advocate the use of ECT in secondary mania, and this case supports the use of ECT in depressive illness secondary to head injury. The effectiveness of ECT on its own has not previously been described in such cases, but reaffirms the findings of Merskey & Woodforde (1972) that ECT might modify the course of secondary depressive illness.

On present evidence we cannot speculate on the neuroanatomical substrates involved in the diverse manifestations of psychotic syndromes. New imaging techniques and post-mortem studies are required to understand the neuropathological basis for these phenomena.

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Organic Manic Syndrome Associated with Advanced Uraemia due to Polycystic Kidney Disease

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Mania secondary to advanced uraemia caused by polycystic disease of the kidneys in a 62-year-old woman involved a complex interplay of physical, psychological and social factors. Psychotherapy and thioridazine led to full recovery.

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Mania in association with uraemia was first reported at the end of the 19th century, but only one case has been documented this century (El-Mallakh *et al*, 1987). These cases are examples of organic manic syndromes, or secondary mania. Other factors, ranging from extracranial to intracranial, may be implicated in the pathogenesis of secondary manias (Krauthammer & Klerman, 1978; Stasiek and Zetin, 1985; Cook *et al*, 1987). In two patients who had been receiving haemodialysis, one developed hypomania following a septic focus (Cooper, 1967) and in the other mania was the presenting feature of a progressive dialysis encephalopathy (Jack *et al*, 1983).

Our case differs from the earlier report with respect to the presence of polycystic kidney disease (PKD) and the sudden eruption of the psychosis during the initial treatment of the chronic renal failure.

Case report

A 62-year-old, previously healthy, married woman presented with a two-week history of weight loss, malaise, dyspnoea, hiccoughing nausea, and vomiting. There was a family history of PKD in her father and one of her brothers but none of mental illness. Examination revealed a normal mental state, sallow skin, a pulse rate of 86/min, blood pressure 105/50 mmHg, jugular venous pressure raised by 3 cm, a pericardial rub, bilateral renal enlargement, and absent ankle jerks. Laboratory investigations were consistent with renal failure. Blood cultures were sterile. Chest X-ray showed cardiomegaly. Abdominal ultrasound revealed multiple cystic areas in grossly enlarged kidneys and smaller cystic lesions in the head of the pancreas and the liver.

Chronic renal failure secondary to PKD was diagnosed. Haemodialysis was instigated, initially with no ill effects, but after the fifth dialysis, nine days later, the patient became overactive, with pressure of speech, elated mood and insomnia. Serum electrolyes were now normal and plasma urea had fallen from 79.8 to 34.4 mmol/l and creatinine from 1.478 to 0.567 mmol/l. By the 11th day she was singing loudly, joking, smiling and talking continuously.

There was no personal history of mental illness. Her birth and early development were normal. She had a good relationship with her three older brothers and her parents. However, after being sexually abused by a neighbour at the age of eight she began truanting and ran away from home on several occasions. At the age of 11 she was placed in a children's home for four years. When she returned home, she worked as a seamstress. At the age of 21 she married. There was considerable marital discord initially and a brief period of separation 15 years ago, but since then she and her husband have worked together in highly successful marital and business partnerships and own their own commercial cleaning company. Both now regard their relationship as close. She had an ectopic pregnancy at the age of 24 and subsequently has been unable to have children. The patient drunk up to two large glasses of beer a day from the age of 26 until two years ago but at no stage have there been any features of alcohol dependence. Premorbidly she was an extravert with a tendency to act impulsively, living for the moment and seldom worrying. She could not recall ever being unhappy for any length of time. Although she worked hard to meet her high standards, she was not obsessional.

At the examination she was overactive, extremely cheerful, continually joking and smiling, and made good eye contact. She was fully orientated in time, place and person and had not been confused. There was marked pressure of speech and it was very difficult to interrupt her conversation. She claimed she felt well, but appeared elated and slightly irritable. She experienced "racing thoughts" and viewed the future with optimism. There were no suicidal thoughts. She also had loosely held ideas (which were not of delusional intensity) that there was 'something strange going on' and that she was responsible for several well publicised tragedies. She also experienced a hypnagogic auditory hallucination telling her to 'live'. On cognitive testing, attention and concentration were impaired but short- and long-term memory were intact. Her level of knowledge was consistent with her educational background and she was of average intelligence. She had limited insight into her condition.

The patient was commenced on thioridazine (100 mg q.i.d.) and supportive psychotherapy. Thrice weekly haemodialysis continued. A computerised tomography (CT) scan of the head revealed no abnormality. An electroencephalogram (EEG) showed a slight generalised excess of fast waves and some theta activity. She recovered from this disturbance of her mental state over the next four weeks and thioridazine was gradually reduced to 50 mg twice a day. After three months she had an indwelling peritoneal catheter inserted under general anaesthetic and has subsequently learned how to manage her own peritoneal dialysis. The thioridazine was gradually withdrawn over four months.

Discussion

The close temporal proximity of the organic insult and the absence of family or personal history of previous psychiatric illness and the late age of onset for the mania made a diagnosis of manic depressive illness (or bipolar disorder) extremely unlikely (Cook *et al*, 1987; Stone, 1989). However, the clinical features of this case did satisfy the criteria for organic manic syndrome (American Psychiatric Association, 1987) or secondary mania (Krauthammer & Klerman, 1978).

Both uraemic encephalopathy and dialysis disequilibrium were considered, but the absence of confusion or disorientation mitigated against the diagnosis of such delirious states (Marshall, 1979). In addition the EEG showed fast activity with some slow waves which may have been induced by thioridazine (Itil, 1974) rather than the typical slowing seen in uraemic encephalopathy (Ginn *et al*, 1975). Dialysis dementia has been a cause of secondary mania (Jack *et al*, 1983), but this condition was excluded as the time course was too short and there was no involvement of higher neurological function.

Although saccular cerebral aneurysms have been reported in association with PKD (Zeier *et al*, 1988), and structural brain disease is a potent cause of late-onset hypomania (Krauthammer & Klerman, 1978; Cook *et al*, 1987), the clinical picture and subsequent investigations did not support a diagnosis of cerebrovascular haemorrhage nor an intracerebral space-occupying lesion.

There were several aetiological factors that should be considered in the pathogenesis of this organic manic syndrome. The disorder was precipitated by chronic renal failure secondary to PKD. It has been suggested that the metabolic derangement of uraemia includes a factor present within the serum that reduces the activity of the sodium-potassium pump within neuronal membranes, leading indirectly to increased neurotransmitter release and the production of mania (El-Mallakh *et al*, 1987). The 'lag period', in our case nine days, between the metabolic disturbance of uraemia and the onset of neuropsychiatric symptoms is well recognised but unexplained (Marshall, 1979). The development of serious physical illness was a major life event. Such events are common before a first manic episode but whether they are causal or precipitant can never be answered absolutely (Ambelas, 1987).

A predisposing factor which might explain why this patient developed mania when so many other patients with uraemia do not was her pre-morbid personality. She showed a tendency to minimise distress, was optimistic, and could never recall being unhappy. This personality structure may have predisposed her to cope with the loss of her renal function by using a manic defence (Klein, 1948). It is interesting that her gradiose ideas had a depressive theme.

Her previous consumption of alcohol, although well above the recommended 'safe limits', had not caused her any significant physical, psychological or social harm. The childhood experience of sexual abuse may have been a contributory factor to her truancy and running away from home but she denied any subsequent problems in her adult life related to the assault.

In summary, the evolution of this organic manic syndrome involved a complex interplay of metabolic, psychological and personality variables. The acute onset of the manic syndrome secondary to the uraemia, the relatively stable pre-morbid personality, the supportive relationship with her husband and the absence of any other psychosocial stressors suggested that the prognosis for her mental health was good providing her physical state remained controlled by peritoneal dialysis.

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Psychosis Following Hydatidiform Mole in a Patient with Recurrent Puerperal Psychosis

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The association of a puerperal psychosis with the removal of a trophoblastic tumour supports the hormonal hypothesis for the aetiology of puerperal psychosis and suggests new lines of investigation into this disorder.

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We present a patient with recurrent puerperal psychosis who developed a similar psychosis following evacuation of a hydatidiform mole.

Case report

The patient was born in 1963. Her childhood and development were normal, although she was a combative and argumentative child. She left school at 16, took up secretarial work and married at 19. She is normally warm and pleasant, but can be abrupt and outspoken and there have been periods of marital conflict. There is no family history of psychiatric disorder and no history of psychiatric or significant medical disorder before her first pregnancy.

In May 1984 she gave birth to a girl. The next day she was tearful, and within a week she became overactive, cleaning the house at 3 a.m. and talking all night. She was also low spirited, suspicious and abusive. She felt people were looking at her and making fun of her, and she accused her husband of harmful intentions. She lost sleep and appetite and her weight fell by 3 kg. She was referred to the psychiatric service two weeks after delivery but denied illness and refused medication. Eight weeks after delivery she was admitted to hospital. She reported auditory hallucinations and doubted if the hospital was truly a hospital. A diagnosis of schizophrenia was made and she was treated with chlorpromazine (100 mg t.d.s.). She improved and was discharged after one month on flupenthixol decanoate (40 mg i.m., fortnightly). At home she felt tired, low, and unable to cope with her household duties, and moved to her mother's house for six months. She then stopped attending for injections, regained her spirits and energy, and returned to live with her husband. There was continued marital friction but the patient remained well for two and a half years.

At 24 she conceived again, but developed vaginal bleeding. An ultrasound scan showed a hydatidiform mole. At approximately 14 weeks' gestation in September 1987 the pregnancy was terminated and the mole was removed. Curettage two weeks later showed no molar tissue, and subsequent urinary chorionic gonadotrophin levels were zero, indicating complete removal. After this event, she described reduced energy and "a terrible feeling of sadness" developing insidiously from the time of termination. Four weeks later she developed otitis media and a friend alleged that her husband had been unfaithful. Her insomnia worsened, and she slept only three hours a night and was restless by day; during the week before her admission she became distressed, tearful, agitated, angry, suspicious and verbally aggressive. She ate little, regurgitated food, and lost weight. Her speech became incoherent. She threatened suicide.

She was admitted to hospital in November 1987. She had lost about 3 kg in a few days. She frequently expressed anxiety that her daughter was being mentally tortured and 'brain-washed' by her mother and doubted if she truly was her daughter. She had ideas of reference, delusional mood, and auditory and visual hallucinations. Throughout her admission she denied illness and refused food and treatment. She was persuaded to accept haloperidol (20 mg daily) to which she developed an acute dystonia. This was changed to dothiepin (150 mg) and thioridazine (100 mg daily) and she was discharged well.

She had her second child in December 1988. Five days later she became irritable and aggressive with labile mood and insomnia. She attacked her mother-in-law, putting her hands around her throat.

She was admitted to the Midland Nerve Hospital. A marked variability of her clinical state was noted with hypomanic phases; she changed from shouting and screaming to laughing and dancing. She improved with haloperidol (7.5 mg daily) and was discharged. She relapsed in February shortly before the onset of her first menstrual period, but recovered again.

Discussion

This patient had three episodes of pregnancy-related illness, the first and third of which were typical of puerperal psychosis, with onset within one week of childbirth, mixed manic and depressive features, and other psychotic phenomena. The second episode was similar to the first, with severe insomnia, anorexia, loss of weight and ideas of reference, but it was less typical of a puerperal psychosis in that the onset was insidious and somewhat delayed, and there were no manic features. In addition there was marital friction, although this was long-standing and not of such a degree as to explain the development of a depressive psychosis.

It has been argued elsewhere (Brockington, 1989) that when patients have two or more pregnancy-related psychiatric illnesses, each episode is probably a manifestation of the same aetiological process; it is therefore of interest that this patient suffered her second episode after evacuation of a hydatidiform mole.

There is no evidence at present that patients with puerperal psychosis develop similar disturbances after miscarriage or termination of normal pregnancies; that is, there is no evidence for a 'post-abortion psychosis' (Brewer, 1977). However, a hydatidiform mole differs from an abortion, since from the hormonal point of view it resembles an advanced pregnancy.

There have been no studies of psychiatric illness due to trophoblastic tumours, but there have been studies of psychological reactions to them: Berkowitz *et al* (1980) sent questionnaires to 44 patients, of whom 30 responded; 18 had been treated with chemotherapy (implying persistent disease) and 12 by evacuation alone. The patients with persistent disease are less relevant, since hormone levels often remain elevated, while complete evacuation leads to a sudden fall. Both groups reported feelings of sadness, and recovery from this mood change took more than a year in 18 of the 30 patients, including five of the 12 with complete evacuation. A larger study of Chinese women (Ngan & Tang, 1986) used personal interviews conducted a year after evacuation of the tumour, and found that 12 of 52 patinets with molar pregnancies described 'sadness' as a sequel. Both these studies were limited in the scope of the psychiatric investigation, not including a full range of psychiatric symptoms or an inquiry into episodes of psychiatric illness. The inquiries were retrospective, often by more than a year, and the numbers were small in relation to the rate for psychosis following childbirth (1 in 500). Within these limitations there was no evidence of psychosis, but many subjects described depression whose severity, duration and type cannot be assessed from the reports.

The importance of this single case is twofold. Firstly, it supports the role of hormonal changes in puerperal psychosis because the most obvious common factor in pregnancy and trophoblastic tumour is the production of large amounts of certain hormones. The hormonal output of hydatidiform moles has been studied by Bagshawe *et al* (1979) and Dawood *et al* (1974; Dawood, 1976). Gonadotrophins are produced to excess, and oestrogens (though not produced by the mole) are produced under the stimulus of gonadotrophins. The output of progesterone by moles can reach very high levels, and the mean level is about the same as in a 32-week pregnancy (Johansson, 1969). Thus all three hormones (gonadotrophins, oestrogens and progesterone) are elevated but to differing extents in different patients, and unfortunately no measurements were made of the output of this particular tumour.

The second reason for reporting this case is that it suggests a new line of inquiry into the possible association of hormone-producing tumours with psychosis. It should be noted that it is not the active tumour which leads to the development of a psychosis, but the successful removal of the tumour, with a precipitous drop in hormone levels. It would be interesting to know whether other hormone-producing tumours, for example arrhenoblastomas and granulosa-cell tumours, are associated with this complication.

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Verapamil in Major (Psychotic) Depression

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Dramatic response of major (psychotic) depression to verapamil in an 82-year-old woman suggests that further study of the use of the drug in affective disorders is required, particularly in the light of a possible aetiological role for calcium.

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The calcium antagonist verapamil has been reported as being effective in the treatment of psychiatric conditions such as affective disorders (Dubovsky & Franks, 1983), panic disorder (Goldstein, 1985), schizophrenia (Price *et al*, 1986) and pre-menstrual syndrome (Price & Giannini, 1986). The majority of reports have been concerned with its role in the treatment of mania, and there has been doubt cast over its efficacy in the treatment of depression (Solomon & Williamson, 1986). We present a case of recovery from a major (psychotic) depressive illness we believe was due to the coincidental administration of verapamil.

Case report

An 82-year-old lady, EC, was admitted to the Elderly Functional Mental Illness Unit at the Royal Liverpool Hospital on 9 December 1988. She presented with a six-week history of lowered mood and increased anxiety associated with marked biological features of depression (early morning wakening; reduced weight, appetite, and concentration; and anhedonia). The onset of these features coincided with a change in her role at the convent at which she resided.

There was no past psychiatric or family history of note. She had been treated for hypertension for 20 years. She had entered a religious order at the age of 25.

On admission she was anxious, although well kempt. She had an expressionless face, but appeared preoccupied and agitated. Her mood was depressed and her speech monotonous, of low volume and reduced spontaneity. The content of her thought was depressive and hypochondriacal. At this time she was thought to be deluded and suicidal. She was fully orientated but showed poor concentration.

She was hypertensive (blood pressure 210/100 mmHg) but there was no evidence of cardiac failure. All other routine investigations proved normal. She had been non-compliant with her medication before admission (however she had previously been on nifedipine (10 mg t.d.s., and tried on flupenthixol, 0.5 mg b.d.).

EC was commenced on fluvoxamine (100 mg daily), which was gradually increased (to 100 mg t.d.s.). She was on this medication for 16 weeks. Her mental state deteriorated and she began suffering from distressing auditory hallucinations. At this point, in view of the serious decline in her mental state, electroconvulsive therapy (ECT) was commenced. Two days after her second treatment, with no improvement in her mental state, she developed a paroxysmal supraventricular tachycardia and cardiac failure. She was transferred to the care of the physicians, her antidepressant was stopped, and she was commenced on verapamil (120 mg t.d.s.) and frusemide.

She returned to the ward, euthymic, two weeks later. She remained well and was discharged three weeks later.

Discussion

The patient received verapamil (120 mg t.d.s.) for the acute management and maintenance therapy of supraventricular tachycardia. Her rapid improvement in mental state over 14 days exceeded even our best expectations had she completed her course of ECT. In these particular circumstances, we felt that the administration of verapamil provided the mechanism for her recovery.

Alternative explanations for such a drastic recovery from a major (psychotic) depression (which the patient described as a miracle) must be considered. The delayed effect of the antidepressant medication is unlikely, as the patient had been on this medication for 16 weeks with a deterioration in her condition. The recovery is unlikely to be the result of two sessions of ECT, particularly as her mental state deterioriated for the two days between her last treatment and transfer. Depressive disorders may spontaneously remit in some cases; however, this is rare, particularly in the elderly. The non-specific placebo effects of a change of medication and transfer to a geriatric ward cannot be excluded. This could have been tested if a placebo had been introduced under double-blind conditions; however, the patient's cardiac conditions precluded this. Acute, severe physical illness is well known, in some cases, to promote a temporary improvement in the mental state of psychotic patients; however, they tend to relapse (mentally) on physical recovery. Further supporting evidence for the role of verapamil in this case is provided by the patient remaining euthymic after four month-follow-up on no psychotropic medication.

Verapamil is a calcium antagonist which acts on the slow calcium channels, and it is widely used in the treatment of supraventricular arrhythmias, angina and hypertension (Singh *et al*, 1978). Previous research has shown that disturbances in extracellular calcium levels can produce various psychiatric illnesses (e.g. Dubovsky & Franks, 1983). In addition it has been widely accepted that calcium has a significant role in the release and synthesis of neurotransmitters (Meltzer, 1986). Dubovsky & Franks (1983) pointed out similarities between lithium and calcium antagonists in that they have both been shown to block calcium influx into brain tissue.

A search of the relevant literature reveals some controversy as to the role, if any, of verapamil in affective disorders. Earlier work (Dubovsky *et al*, 1982; Giannini, 1984) tends to point to its efficacy in mania, and reports on its role in depression have been contradictory. Solomon & Williamson (1986) cast doubt over its efficacy, whereas Pollack & Rosenbaum (1987) report a case of recurrent unipolar depression in which verapamil was used for both prophylaxis and treatment of the acute episode.

Thus the role of verapamil in affective disorders is far from proven, although a body of clinical findings is now building, suggesting its efficacy in some cases. Its effects on calcium metabolism and, through that, neurotransmitter release and synthesis, along with apparently similar clinical indications to those for lithium, may shed further light on the biochemical aetiology of affective disorders. It is certainly worthy, in our opinion, of further research.

Acknowledgements

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A Steroid Stupor in a Surgical Ward

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In the development and management of a steroid-induced stupor, in a 17-year-old man, the dose and route of administration of steroid medication were felt to be important aetiological factors. A co-ordinated plan of management involving the physician, surgeon and psychiatrist is needed in such cases.

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Since the introduction of steroid medication in 1950 there have been many reports of associated psychiatric symptoms, and these have increased as the therapeutic indications for the medication have broadened. Rome & Braceland (1952) described four grades of psychiatric response to steroids, ranging from mild euphoria to psychoses. The frequency of steroid-induced psychiatric disorders increases with the daily dose of medication, ranging from 4.2% of those taking 40–80 mg prednisone or equivalent to 18% of those taking more than 80 mg (Boston

Collaborative Drug Surveillance Program, 1972). The descriptive psychopathology of steroid-induced psychosis is frequently an admixture of affective, schizophreniform, and organic symptoms (Clark *et al*, 1952). In a study of 14 patients, previous episodes of steroid psychoses, pre-morbid personality and previous psychiatric history did not predict the development of a current steroid psychotic disorder (Hall *et al*, 1979).

The management of patients with steroid psychoses is particularly difficult because, in addition to the psychiatric disorder, there is the physical illness for which the steroids were initially prescribed. We describe a particularly difficult case of a young man with ulcerative colitis who developed a steroid-induced stupor.

Case report

The patient, a 17-year-old student, developed diarrhoea with blood and pus in December 1987. Sigmoidoscopy revealed a mild inflammation of the rectum and distal colon; the biopsy report described a non-specific proctitis. He was admitted in March 1988 with increased frequency of blood-stained motions associated with left-sided abdominal pain. A double-contrast barium enema revealed a mild pan colitis extending to the caecum; there was not toxic dilatation of the colon. He was treated with metronidazole, mesalazine and steroid enemas with good response.

Three weeks later there was a serious relapse of his colitis, with more than 20 bowel motions per day. He was commenced on hydrocortisone (100 mg i.v. q.i.d.) during which time he had a short-lived acute anxiety episode. His bowel symptoms settled within two days and his medication was changed to oral prednisone (20 mg t.i.d.) on which he remained for six days until a moderate relapse (6–8 motions daily) of the colitis necessitated the reintroduction of hydrocortisone (100 mg i.v. q.i.d.). After a further six days on this regime he became increasingly agitated and frightened, and he quite unexpectedly made a serious suicide attempt. At this stage the bowel symptoms had improved again and his medication was changed to adrenocorticotrophic hormone (ACTH) (40 units i.m. daily) which was gradually decreased and stopped over the next ten days. However, his mental state continued to deteriorate, with the rapid development of a catatonic stupor characterised by mutism, waxy flexibility, and *mitgehen*. There was no personal or family history of psychiatric illness. A computerised tomography (CT) scan of the brain was normal and an electroencephalography (EEG) revealed mild, diffuse, slow-wave activity.

He was commenced on haloperidol (5 mg b.d.) which was changed to chlorpromazine (50 mg b.d. and 100 mg *nocte*) because of extrapyramidal symptoms. Over four weeks his stupor improved but moderate psychomotor retardation remained. His thought content was now more accessible: he was frightened and perplexed and had delusions of passivity and third-person auditory hallucinations. This later changed to depressed mood with nihilistic delusions and suicidal ideas. It was decided at this stage to proceed with electroconvulsive therapy (ECT) because of the slow response to neuroleptic medication and the intensity of his psychotic symptoms. He made an excellent response to a course of five treatments.

Following a conference involving the surgeons, physician and psychiatrists, it was decided to proceed with a surgical approach for the management of the colitis because of the extent of the inflammation and the severity of the recent exacerbation of the colitis. The steroid psychosis did not have a major influence on the decision except that non-operative therapy of his colitis would have presented a dilemma at his next acute exacerbation. The surgical procedure consisted of two stages, initially a total colectomy, ileo-anal pouch anastamosis and temporary ileostomy, followed six weeks later by a reversal of the ileostomy. This operation was favoured because of the psychological benefits to a young man of not having a permanent ileostomy. One year later he was doing very well, had made an excellent social adjustment, and was pursuing an academic career.

Discussion

Two factors may explain the severity of the steroid-induced stupor described. The high dose of hydrocortisone was equivalent to 80 mg prednisone daily and this would have been the major aetiological factor in precipitating our patient's acute psychosis. The intravenous route would also have contributed to the severity of the symptoms, as the peak plasma level of steroid medication is thought to be related to the development of psychiatric symptoms (Glynne-Jones *et al*, 1986; Tomson & Venning, 1987). It is interesting that although the clinical picture in steroid psychosis frequently has an affective component, as in our patient, antidepressants are likely to make the symptoms worse, and neuroleptic medication should be used (Hall *et al*, 1978; Malinow & Dorsch, 1985). The change of medication from intravenous hydrocortisone to intramuscular ACTH reflected an attempt to minimise peak plasma levels of steroid, but clearly this did not prevent progression of our patient's symptoms to a full-blown stuporose state. The dose of ACTH used was equivalent to a smaller dose of hydrocortisone than had been given to this patient initially, but accurate comparisons were not possible since cortisol levels while on ACTH were not measured. Our patient's mental state was not likely to reflect a toxic confusional state, since his colitis was responding to treatment, his electrolytes were normal at all times, and there was no evidence of infection.

In the management of these difficult cases treatment of the physical illness must take priority, as stopping the steroid medication is not an essential prerequisite for the treatment of a steroid psychosis

(Glynne-Jones *et al*, 1986). Clinicians should not be deterred from prescribing steroid medication if indicated for a patient who has a previous psychiatric history (Lewis & Fleminger, 1954; Alcena & Alexopoulos, 1985). However, with any patient on high-dose steroid medication the clinician should take good note of any behavioural or psychological change reported by a relative or nursing staff, as this may herald the onset of a psychosis. The majority of steroid psychoses have an onset within five days of commencing medication; however, this may vary up to four weeks (Kaufman *et al*, 1981). Although our patient had been on steroids for two weeks, the *onset* of the psychosis seemed to be more related to the intensive intravenous course of hydrocortisone.

In cases of steroid psychoses a planned multidisciplinary approach is important, particularly in the identification of priorities for treatment and the formulation of both short-term and long-term plans of management.

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Depression-Dependent Erotomanic Delusions in a Mentally Handicapped Woman

M. GHAZIUDDIN and L. TSAI

Erotomanic delusions occurred in a depressed mentally handicapped woman. Although erotomania is generally classified with paranoid disorders, in people with mental handicap, depression may be the more commonly associated illness.

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Erotomania is a rare psychiatric condition in which the patient, usually a woman, firmly believes that a person, usually of higher status, is in love with her (Segal, 1989). It can be classified into two forms: a primary type, in which no other psychiatric condition coexists, and a secondary type, which is associated with other psychiatric disorders (Enoch & Trethowan, 1979). Literature suggests that it has generally been regarded as closer to schizophrenia and paranoid disorders than to mood disorders. Thus, it is classified as a type of delusional (paranoid) disorder in DSM–III–R (Kendler *et al*, 1989), and is said to respond in its 'pure' form to pimozide, a diphenylbutylpiperidine antipsychotic (Munro *et al*, 1985). However, some recent studies have suggested that features of major affective disorder may be common in these patients. For example, Signer & Swinson (1987) reported two cases of erotomania in women with bipolar affective disorder arising or persisting during periods of euthymia. While erotomania has been described in a variety of conditions ranging from amphetamine abuse and alcoholism to organic brain syndrome (Lovett-Doust & Christie, 1978; El Gaddel, 1989), its association with mental handicap appears to be uncommon.

We report a mentally handicapped woman who presented with depression-dependent erotomanic delusions. To the best of our knowledge, erotomania in the setting of an affective disorder has not been described in the mentally handicapped population.

Case report

M is a 25-year-old mentally handicapped woman who lives in a group home with five other handicapped clients. She has basic verbal and self-care skills. She has some spasticity of her lower limbs, but is able to walk without support. She has a social age of six years and three months (Vineland Social Maturity Scale). A psychological assessment done three years ago estimated her performance to fall within the moderately handicapped range. Although the exact aetiology of her handicap is unclear, she carries a diagnosis of spastic/athetoid cerebral palsy.

She was referred to our clinic with a six- to nine-month history of sexually disinhibited behaviour (including masturbating in public) and aggression and frequent crying. Although she had always had occasional tantrums, her crying spells had become more frequent and prolonged. Her sleep and appetite were normal although her self-biting had increased. She thought that she was disliked by the other clients and wondered what it would be like to run away. She started talking about death and a few months later (two months before this referral) she was reportedly found with a piece of cloth tied around her neck.

Although the exact onset of erotomania is not clear, it is reported that sometime after she became depressed, she started believing that the head teacher of her school was in love with her. She claimed that although he was a married man with children, he wanted to have sex with her. This belief gradually strengthened and she was soon writing love letters to him, occasionally insisting on seeing him at school. Her doctor prescribed imipramine (20 mg nocte, increased to 30 mg after five months). At the time of the present referral, she had been on 30 mg for a month.

There is no family or past history of psychiatric illness although her father is said to have abused alcohol. Her parents divorced when she was 15 years old. Since then, her mother has moved while her father still lives locally and keeps in occasional contact with her. Since their divorce, M has been in at least three group homes; she came to her present home five years ago. There is no other significant history available.

She was a sad, bespectacled, Caucasian woman, who looked younger than her age. Despite her gentle rocking, she was reasonably co-operative and relaxed. She had a mild stutter which did not seem to interfere much with her speech, and she could understand the questions. She looked depressed and admitted that she felt sad and cried often. She firmly believed that the head teacher was in love with her, that he was the one who initiated the relationship, and that he wanted to have sex with her. She could not explain why she held this belief. Apart from this, no other abnormality was noted. Her score on the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) was at least 16, although some of the items could not be rated because of her intellectual deficit. After doing a baseline electrocardiogram, she was reassessed after two weeks and her imipramine was increased to 75 mg at night. Six weeks later, she appeared much brighter. She was crying less often and also sleeping better. Previously, she used to write at least four letters every week to the head teacher, but now she had not written any letter in the two weeks before the follow-up appointment. When asked about the head teacher she remarked: "He is a married man; he has his own family." It was clear from the staff reports that the abnormal belief was almost gone, although not entirely. Her HRSD score on this occasion was seven.

When seen six weeks later, she was reported to have deteriorated. She was crying more often and also interacting less with the other clients. In addition, she had again started talking about the head teacher, how much he loved her and wanted to sleep with her. She looked depressed, and said that the staff did not like her and that she felt bad about herself. Her HRSD score increased to 14. While the exact cause of her deterioration was unclear, three events seemed significant: the departure of a female member of staff, to whom she was much attached; an angry visit by her father, who was reportedly drunk at the time of the visit; and the closure of her school for the summer break. Suitable environmental changes were suggested, and her imipramine was increased to 100 mg at night.

On her next appointment, two months later, her mood was much improved. She was sleeping and eating better, and staff thought that she looked 'wonderful'. At the same time, there was almost no mention of the head teacher and on direct questioning, she remarked that "he is a married man with his own family . . ."

M has been followed up for about eight months since her initial presentation. Her mood remains stable. She does not write to her head teacher anymore, nor has she recently talked about his love for her. She continues to stay on the same dose of imipramine (100 mg at night).

Discussion

This patient presented with typical features of erotomania. She had a firm belief that her head teacher, a man higher in social status than her, loved her and wanted to have sex with her. The fact that she wrote to him also suggests the intensity of her abnormal belief. While the exact onset of her depression cannot be commented upon with certainty, the information available strongly suggests that her depression preceded her erotomanic delusions. In addition, her delusions seemed to be depression-dependent – they seemed to wax and wane according to the intensity of her depression. The correlation with the scores on the HRSD seems to support this view.

Very few reports have been published that describe erotomanic delusions in mentally handicapped people. This may, in part, reflect the difficulty involved in diagnosing mental illness in this population. Thus, Greyson & Akhtar (1977) described erotomania in a 46-year-old mentally handicapped man; it was not clear whether he suffered from clinical depression or whether he was tried on antidepressants. Collacott (1987) described two cases: the first responded to phenothiazines and was said to have 'pure' erotomania; the second failed to respond to phenothiazines, antidepressants, or butyrophenones – her likely psychiatric diagnosis was schizophrenia. The present case suggests the need to be aware of the possibility of depression in such cases and also highlights the efficacy of antidepressants when used in therapeutic doses.

Various psychological theories have been proposed to explain the aetiology of erotomania. Freud viewed it as a defence against homosexuality while others have argued that the main function of erotomania is to serve narcissistic needs, especially in those who lead lonely and socially empty lives (Segal, 1989). The above patient's history seems to support this view. Abandoned by her family, she led an essentially lonely life. She had no friends or hobbies. It can be speculated that the belief that she was loved and wanted served to compensate for the disappointments of her life. To what extent her low intelligence and other factors, both psychosocial and biological, could have contributed to her symptom formation is not clear. Fenichel (1945) suggested that the important factor in erotomanic psychosis was imminent object loss, which the patient defended against by clinging to a love object. Based on this assumption, it has been claimed that mentally handicapped people are vulnerable to erotomanic delusions because of the many losses they sustain (Collacott, 1987). Since loss events have an association with depression (Paykel, 1982), it is possible that erotomanic delusions in mental handicap are more commonly associated with depression than with other psychiatric disorders. It may be useful to explore this issue further because of its obvious clinical relevance.

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Editor: Ian Pullen

Contents: CPK in NMS/Understanding Capgras syndrome/Eating disorder in Asian women/Transsexualism/Carbamazepine in alcohol withdrawal/Culture as a confounding variable?/Buspirone in detoxification/Is psychiatric training still improving?/Are British psychiatrists racist?/ECT following clozapine/Buspirone-induced mania/Educating the psychiatrist of the 21st century

CPK in NMS

SIR: We are concerned at the inappropriate emphasis being placed on creatinine phosphokinase (CPK) estimation in the diagnosis of neuroleptic malignant syndrome (NMS) and in its differential diagnosis from other disorders. We concur with Gratz & Simpson (*Journal*, October 1990, **157**, 617–618) that elevated levels of CPK may occur as a result of untreated rigidity and wish to emphasise that this need not necessarily be induced by neuroleptics. The recent case report by Dalkin & Lee (*Journal*, September 1990, **157**, 437–438) states that their diagnosis of NMS "rests" on the raised CPK, and they dismiss the possibility that this may be the result of severe dystonia because "there are no reports in the literature of neuroleptic-induced dystonic reactions causing raised CPK levels". Levenson (1985) includes elevated CPK as one of three major criteria of diagnostic importance in NMS and implies that raised CPK differentiates NMS from catatonia. Some other authors have, however, been more cautious about using CPK measurements in such a definitive fashion. Adityanjee *et al* (1988) stress that, when diagnosing NMS, CPK and leucocytosis should be used only in support of the clinical findings.

We report a case of catatonia in which CPK estimates reflected the clinical picture.

Case report: A 19-year-old single woman with no past psychiatric or medical history of note developed an acute paranoid psychosis with progressive motor disturbances including stereotyped rocking, facial grimacing and withdrawal, followed by stupor with incontinence. Four days after the onset she became rigid, postured for long periods and displayed waxy flexibility and resistance. This was interrupted by short periods of excited motor behaviour and high pitched squeals. On admission all blood results including full blood count, erythrocyte sedimentation rate and CPK were normal, as was a urine drug screen. As the catatonic movements became more severe, her CPK rose reaching a maximum of 1220 iu/l six days after admission. Twelve days after admission electroconvulsive therapy was commenced because of her continued catatonic state with no oral food or fluid intake. Her condition responded rapidly and after seven treatments she was completely recovered. At one week following recovery, CPK had returned to within normal limits (0–60 iu/l). At no time before or during this patient's illness did she receive neuroleptic or other psychotropic medication.

CPK is found in muscle and brain tissue and provides a measure of tissue catabolism. There are three isoenzymes, of which CPK MM most closely reflects muscle activity and integrity. CPK MM may be raised by skeletal muscle damage (including intramuscular injections, exercise, trauma and massage) and by some drugs including succinylcholine, alcohol and lithium. It is also raised in malignant hyperpyrexia, hypothermia, coma, infectious diseases involving muscle, convulsions, hypothyroidism and muscular rigidity. It may be raised in extreme muscular activity due to agitation as seen in psychotic patients (Ford, 1987).

Our case is a clear demonstration that CPK may be elevated when catatonic symptoms occur in the absence of NMS. We believe that, when diagnosing NMS, it is inappropriate to put undue emphasis on the presence of elevated CPK which is, after all, simply a non-specific indicator of muscular catabolism.

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Understanding Capgras syndrome

SIR: In Anderson's comments (*Journal*, October 1990, **157**, 622–623) on Rastogi's article (*Journal*, June 1990, **156**, 883–884) he says that he is "disappointed to see once again the delusion of inanimate doubles explained away by psychological symbolism". I write to underline this view.

In his letter Dr Anderson also refers to my article (Lipkin, 1988). While I ended this by saying "it is the recommendation of the author that all patients presenting with a Capgras syndrome, whether obviously psychotic or not and whether there be evidence of early dementia or not, be investigated by electrophysiological and brain-imaging techniques", I should like to add a further comment. In my paper I noted that the patient discussed "was referred for second opinion by her consultant psychiatrist...". It is perhaps not insignificant that the referring consultant psychiatrist was a psychoanalyst. Important aspects of that particular patient's life gave some credence to the notion that her misidentification of her second husband was the manifestation of the psychological mechanism of 'denial'. Psychological treatment proved fruitless (not necessarily because 'denial' was not in operation), because the underlying problem was an organic one.

Therefore I share the view of many others, including Dr Anderson, that it is necessary to be aware that the Capgras syndrome and Capgras-like phenomena frequently have an organic base and that a preoccupation with putative psychodynamic formulations are not necessarily in the best interests of our patients.

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Eating disorder in Asian women

SIR: I read with interest the paper by Dolan *et al* (*Journal*, October 1990, **157**, 523–528) on the eating attitudes of British women from three different backgrounds (Caucasian, Afro-Caribbean and Asian). Despite their much lower referral rate for psychiatric treatment, Asian women were found to have significantly more disordered eating attitudes which, unlike Caucasian women, were not correlated with a similar degree of anxiety and depression. The paper demonstrated the different relationships between disordered eating attitudes (as shown by the English Eating Attitude Test; EAT) and anxiety and depression, and the implications on clinical presentation of eating disorders. However, a few points deserve mention.

Firstly, the mean age of the Asian sample is high (31.7 years) and not ideal for studying eating attitudes related to acculturation. No information was given as to whether the subjects were immigrants or second-generation British Asians. Previous studies (e.g. Mumford & Whitehouse, 1988) found disordered eating behaviour to be unexpectedly common in second-generation young British Asians (aged 14–16) who grew up with Western notions of slimness and dieting preoccupations. Social class, a known confounding variable in the EAT, is not mentioned.

Secondly, attitudes toward eating and body shape indeed vary greatly. Anorectic patients from different cultures may manifest different clinical patterns (Lee, 1989), and the use of Western instruments to assess eating attitude and body image in non-Western groups is fraught with controversies. More than 10% of the Asians did not attempt the questionnaire because of difficulty in reading English, and one wonders how many of the rest (43 of them, who constituted only 8.3% of the total sample and were understandably 'precious' subjects) had linguistic and conceptual problems (their education was not mentioned) which might lead to misinterpretations of the EAT and anomalous answers (King & Bhugra, 1989). This is especially relevant as Asian subjects did not have much anxiety and depression despite high EAT scores. The EAT has been factor-analysed to yield three subscales which may predict clinical patterns and outcome of eating disorders (Garner *et al*, 1982). It may be instructive for the authors to analyse accordingly for any meaningful (or chaotic) patterns instead of using the global score alone.

Thirdly, the lack of association of disordered eating attitudes with depression paralleled a recent Taiwan study of Chinese patients with eating disorders where, in contrast to Western findings, not one case of major depression was identified (Tseng *et al*, 1989). But the authors' assertion that "some degree of emotional distress is necessary to precipitate a request for help" is only right with respect to *mental health professionals*, as Chinese anorectic patients may seek help from physicians (for bloating and emaciation), gynaecologists (for amenorrhoea) or even herbalists (for energy imbalance). They rightly commented that doctors might not recognise eating disorders in non-white women, because the diagnosis of eating disorder involves the dialectical process of illness negotiation between *both* the patient and the doctor (Swartz, 1987). In particular, doctors now in medical practice are likely to have been taught as medical students that eating disorder is a rare condition of the upper-class, white family. *Re-education* is obviously helpful, as this seems so inaccurate today.

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Transsexualism

Sir: We wish to comment upon the two contributions to the literature on transsexualism (Mate-Kole *et al, Journal*, August 1990, **157**, 261–264; Burns *et al, Journal*, August 1990, **157**, 265–268). The first study is concerned with the outcome of gender reassignment and compares operated and waiting list patients. It is useful to present composite measures in terms of social activity and a self-assessment scale measuring neurotic symptoms. From these measures a generally improved quality of life may be discerned although the most important questions are left unanswered; these concern the patients' attitudes to the whole process of gender reassignment and the satisfaction, in retrospect, of having undergone the arduous procedures of approximation to the gender role requested. The matter of outcome assessment of this extraordinary intervention, conducted at the earnest request of the patient, requires greater attention than it has currently received, and assessments must be conducted by persons who are independent of the clinical team staff and 'neutral' in attitude toward the procedure of gender reassignment (Abramowitz, 1986; Snaith, 1987).

A small outcome study of our own, conducted with respect to these criteria, was reported briefly (Snaith, 1990). This gave us confidence to continue with the work but clearly more extensive studies are required to guide clinicians in these difficult decisions. We need to know more about the fate of those requesting gender reassignment whose requests are refused by gender identity clinic staff. At the time of rejection of application many patients appear to be very distraught but it may be that a number can later accept the grounds for refusal and lead more contented lives in pursuit of realistic aims.

The second study calls for further study of the phenomenology of transsexualism and the relation of these factors to outcome. This also is an important area of study. It is probable that whether or not the person fits the criteria of the DSM–III is of lesser importance than whether the person can live in the opposite gender role and be accepted in that role by family, friends and employers. The general stability of personality and ego strength are not assessed by the DSM but are vital factors in outcome. The care and pace at which a person is guided through a gender reassignment programme and the supervision of all aspects of this complicated procedure must certainly have a major effect upon the eventual outcome, relief of distress and improved quality of life which are the ultimate criteria on which reassignment procedures must be judged.

This close supervision can only be provided by a regional gender identity clinic team. The metropolitan centres at Charing Cross Hospital and the Maudsley Hospital offer a considerable service by accepting referrals without geographical restrictions but, for all their experience and excellence, they cannot provide the close social, psychological and endocrinological supervision to people living at a distance of some hundreds of miles; nor can they be immediately on hand to deal with the sometimes severe post-operative complications.

Although the nature of transsexualism remains elusive it is a human condition which is not likely to disappear and people will be requesting the services of experienced teams for many years to come. It is our experience that a satisfactory service can be established on a regional basis within the National Health Service. As regards private sector services these have the inherent danger that patients may purchase what they initially believe to be in their interests but which may end in disaster.

Transsexualism is listed in the classificatory systems as a psychiatric disorder. Whether or not this is correct may be debated but Gender Identity Clinics should be established under the supervision of psychiatrists who have the experience to assess stability of personality and to detect latent psychiatric disorder which would contraindicate gender reassignment. The Royal College of Psychiatrists should now conduct a national enquiry of clinic practice in this area and contract to set up some general guidelines as to future practice.

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Carbamazepine in alcohol withdrawal

Sir: The article by Glue & Nutt (*Journal*, October 1990, **157**, 481–490) on overexcitement and disinhibition was both interesting and informative. It did however omit a discussion of carbamazepine in the treatment section.

Carbamazepine has been shown in a controlled trial (Malcom *et al*, 1989) to be of equal efficacy to oxazepam in reducing the symptoms of alcohol withdrawal. The authors postulated that this was due to its 'antikindling' effects, although its action on presynaptic adenosine receptors may also be important (Durcan & Morgan, 1990). In addition, with an increasing emphasis on out-patient detoxification programmes (Collins *et al*, 1990), carbamazepine has the advantage of a low potential for abuse or dependency.

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Culture as a confounding variable?

Sir: In their study of thought disorder in schizophrenics, manic–depressives and major depressives, Cutting & Murphy (*Journal*, September 1990, **157**, 355–358) were careful to compare their groups for IQ, age, sex and attentional factors. The study involved a judgement of the subjects' answers to multiple choice questions regarding social knowledge about their culture and general knowledge about the state of the world. It is a shame that the care taken to examine psychological differences is not matched by an equal care to examine social differences between the groups.

The important influence of culture in psychiatry is increasingly recognised and debated (see Leff, 1990; Littlewood, 1990). No mention is made in Drs Cutting & Murphy's study of the cultural background of the groups. This will surely have considerable influence on their judgement of a subject's knowledge of his or her own culture! If this questionnaire is to be of general use its validity across different cultures should be tested.

The difficulty in deciding which are social factors is demonstrated by the *post hoc* change of category of one question. From the 'non-social' category the answer to the question, 'What is the age of the oldest person in Britain?' will surely be influenced by the social experience of the respondent. If they come from a Nepalese culture this will be of a less elderly population than a respondent from another culture. The influences of culture must not be overlooked.

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Buspirone in detoxification

Sir: I wish to comment on Ashton *et al*'s study (*Journal*, August 1990, **157**, 232–238).

The practice of prescribing additional drugs to aid detoxification in drug-dependent individuals is a controversial issue. The addition of one anxiolytic agent (busipirone) to aid withdrawal from another anxiolytic agent (diazepam) appears contradictory, especially when buspirone's data sheet specifically states that it will not block the withdrawal syndrome often seen with cessation of diazepam therapy. Furthermore it is an ideology that pushes the more immediate question of motivation into the background.

The authors argue that in certain circumstances such as benzodiazepine withdrawal, buspirone displays antagonist rather than agonist effects at the 5-HT1A receptor sites. This is a gross oversimplification of the pharmacodynamic profile of chronically anxious, withdrawing, benzodiazepine-dependent individuals on buspirone. No mention is made of possible interactions causing reduced dorsal raphe nucleus activity and hence reduced behavioural inhibition (Eison *et al*, 1983). In contrast to the benzodiazepines, buspirone increases locus coerulus firing (Sanghera *et al*, 1985). Studies report subjective and objective improvement in alertness and concentration and reduced confusion in patients taking buspirone compared with those taking benzodiazepines (Schweizer *et al*, 1986). It is not known, however, whether the combination of buspirone and a benzodiazepine produces changes in cognition. Thus an isolated discussion of 5-HT1A receptor effects does not do justice to what is known of both buspirone and diazepam.

Any study, no matter how well designed, as was the case here, can suffer from unforeseen errors. Randomisation does not guarantee equivalence and in this study there is no avoiding the fact that the group assigned buspirone were initially more anxious. In retrospect a stratified randomisation might have been a better design. It is my opinion that the more anxious the individual the slower their withdrawal should be. It was a small study and I would have appreciated the inclusion of confidence intervals to provide me with a range of uncertainty (Gardner & Altman, 1990). It has been suggested that weekly rating intervals are inadequate for detecting the full range of relapse, rebound and withdrawal phenomena because symptom increases may be transient (several days) and go undetected with weekly assessments (Rickels *et al*, 1986).

Out of 23 patients, 17 had a good outcome in the study, remaining off benzodiazepines at six and 12 months and here the authors should be congratulated. However, considering the high dropout rate for the buspirone group I am tempted to believe that the better outcome in the control group could in part be due to the authors' continued contact and rapport with these patients, especially since all the buspirone-treated patients who remained in the study achieved a similar good result.

In conclusion, when considering data from studies of adjunctive pharmacotherapy in withdrawal states, it is important to have a conceptual framework that allows specific drug–drug interactions to be viewed as occurring in a dynamic nervous system that has functional plasticity, multiple systems of parallel processing and complex heirarchical association mechanisms. In this way tentative statements of drug agonistic or antagonistic effects can be more realistically appreciated.

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Is psychiatric training still improving?

SIR: We read Brook's article "Is psychiatric training still improving" (*Journal*, September 1990, **157**, 335–338) with interest. As visiting psychiatric trainees from different countries, we hope to learn from psychiatric training in Britain.

The opinions of newly appointed consultants about their training provided the basis of Dr Brook's study. Unfortunately the opinions expressed as to satisfaction were retrospective, with the possibility of a personal 'halo' bias giving a more favourable view of their training. Also respondents were those ex-trainees who have successfully obtained consultant posts; what of the others who failed at this hurdle?

Dr Brook stated that statistical analysis would be inappropriate for 'opinions'. We would disagree. Statistical analysis is particularly important when one is dealing with such data if they are to be useful. Although Dr Brook reiterated that the results reflected opinions about training and that what may have been considered satisfactory ten years ago may not be considered satisfactory today, we feel that the results of the 1982 and 1987 surveys should be subjected to some form of statistical analysis if they are to be compared. Dr Brook's assessments of the results are subjective and hence debatable.

We welcome Dr Brook's suggestions that trainees rate their experiences at intervals as this would remove the bias due to retrospective recall; and trainees should continue to identify inadequacies in their training.

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Are British psychiatrists racist?

Sir: Lewis *et al*'s study of racial bias in psychiatric perception (*Journal*, September 1990, **157**, 410–415) is valuable. While bias may, of course, be elicited with one particular vignette and not with another, it is significant that neither their study nor a similar one of mine (*Journal*, September 1990, **157**, 451–452) found any greater tendency to diagnose schizophrenia among black patients when stated ethnicity was the sole variable. A problem remains, however, of the relationship between the leisurely rating of vignettes and the actual clinical decision-making (in which the differential perception of dangerousness, which Drs Lewis *et al* find, may well be rationalised subsequently through a diagnosis of schizophrenia, on the way to custodial and neuroleptic treatment).

Acute psychotic reactions were rated marginally more frequently in the Afro-Caribbean vignettes than in those of the whites, but I am not certain that Lipsedge and I are as responsible for this as they gently hint. Take the characteristics identified by their respondents, especially in the black vignette: duration less than three months, risk of violence to staff, neuroleptic treatment indicated, and to be charged with criminal damage. Only the first – 'acute' course – corresponds to anything in the clinical profile we derived in the paper (Littlewood & Lipsedge, 1981*b*) accompanying the one they cite (1981*a*). Our profile was based on those patients clinically given a diagnosis of schizophrenia who did not have core symptoms as rated by the research Present State Examination. Furthermore we specified acute 'psychosis' as a stay in hospital of less than three weeks, not three months. In their study, however, schizophrenia is preferentially diagnosed among whites. If there has indeed been a switch in diagnostic preferences among British psychiatrists since 1981 from schizophrenia to acute psychotic reactions for black patients, as a consequence of our paper, this does not seem to have been reflected in recent epidemiological studies. Our use of the term 'acute' was directly related to a Jasperian notion of 'reactivity', not just to duration of the illness. The idea of psychotic reactions of short duration among Afro-Caribbean patients in Britain had been around since at least Tewfik & Okasha's (1965) study. In its stereotyped form this category was criticised for its racism by Lipsedge and myself in a book (Littlewood & Lipsedge, 1982) arguably better known than our papers.

The rather more difficult question is· how and why do psychiatrists use stereotyped judgements? Are they indeed something specific to a psychiatric theory still embedded in imperial fancy, or is it that psychiatric care simply replicates the social power and prejudice located outside medicine? My own vignette study, which showed that medical students before and after they studied psychiatry, and psychiatrists themselves, all had similar perceptions, argues against the ideological power of specific psychiatric theories in themselves.

Both vignette studies would seem to dispute such a power. If transcultural psychiatry in Britain has correctly shifted from its exclusive focus on the black patient to examine the role of the white psychiatrist, we have to be prepared to look at the particular social context of power within which psychiatry operates, which determines the perceptions and responses of both patient and doctor, and their interaction.

Such studies would hardly be independent of an understanding of racism in its wider economic, ideological and coercive forms.

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ECT following clozapine

Sir: The safety of electroconvulsive therapy (ECT) following clozapine therapy has not been documented. The potential for spontaneous seizure phenomena is of particular concern in light of

clozapine's suspected epileptogenic properties (Heerlen & Kunze, 1979; Juul Povlsen *et al*, 1985). Herein, we report on one patient who developed spontaneous (tardive) seizures following only one electroconvulsive therapy treatment soon after clozapine discontinuation.

Case report: A 26-year-old man with a four-year history of chronic paranoid schizophrenia received up to 800 mg per day of clozapine with limited response as extreme anxiety, fearfulness and paranoia continued to impair his ability to function. At the time that ECT was recommended the patient was receiving clozapine (800 mg), propranolol (60 mg) and diazepam (20 mg) daily, which were then tapered over 14 days and discontinued, with the exception of diazepam which was reduced to 5 mg daily 72 hours before his first ECT session. The patient received bilateral ECT four days after his last dose of clozapine. Seizure duration monitored by two-lead electroencephalography (EEG) was 123 seconds. Recovery was remarkable for significant post-ictal confusion. The patient had two spontaneous *grand mal* seizures witnessed by staff on days four and six following this first and only ECT treatment. An EEG performed on day five after the first seizure and before the prescription of phenytoin was remarkable for mild defused slowing, with a 'questionable' focal abnormality in the right frontotemporal region. Non-contrast computerised tomography and magnetic resonance imaging were normal. The patient was ultimately discharged on maintenance phenytoin and clozapine with minimal improvement in his mental status, but no further seizure activity.

The inferences that can be drawn from this care are, however, unclear. We cannot rule out the possibility that clozapine stopped only four days before ECT in some way facilitated this patient's spontaneous seizures. However, other explanations are equally plausible. The patient had been on long-term benzodiazepine treatment which was reduced from 20 mg to 5 mg over two weeks; this may have been a contributing factor. It is also impossible to tell from an individual case whether this patient's seizure activity is significant in light of the reported one in 500 incidence of tardive seizure phenomena following ECT (Fink, 1977).

As a practical guide, given the suggestion of increased epileptogenic activity with the atypical neuroleptic clozapine, clinicians are advised to permit a drug-free period of 7–10 days following clozapine discontinuation before starting ECT. Theoretical risks of spontaneous seizure activity and prolonged seizure duration are raised which merit further study.

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Buspirone-induced mania

SIR: Buspirone, with its limited liability for abuse (Griffith *et al*, 1986), and lack of withdrawal effects (Tyrer *et al*, 1985), might be considered the drug of choice in the treatment of anxiety in patients with a history of alcohol or drug abuse. Such a patient, who developed mania after two weeks on buspirone, is presented.

Case report: A 28-year-old single man began abusing alcohol and heroin at 14 years of age. From 1984 onward he binged sporadically on alcohol, but remained off heroin. He presented for treatment in November 1989, with severe symptoms of anxiety. He was commenced on buspirone (10 mg b.d.), and also agreed to take disulfiram (400 mg daily). He took the buspirone regularly for two weeks, but took the disulfiram only intermittently. He drank on occasion during this period, but denies any other drug abuse. While on buspirone he described a 'floating feeling', and noted his thoughts going faster. Over the next few weeks he developed pressure of speech, flight of ideas, irritability, elated mood, and overactivity. There was no clouding of consciousness and he denied perceptional disturbances. Physical examination was normal. A drug screen was negative. The patient required high-dose neuroleptic medication to which he responded slowly. Neither he nor his family have any history of affective disorders.

To date there has been three published reports of buspirone causing mania. Two of these (Liegghio & Yeragani, 1988; McDaniel *et al*, 1990) involved patients who already had a diagnosis of bipolar disorder. In both, the introduction of buspirone precipitated a manic swing. In the third (Price & Bielefeld, 1989) a 38-year-old man with resistant depression and anxiety became hypomanic on the two separate occasions that buspirone was introduced.

Buspirone is thought to exert its effect by interaction with 5-HT1A receptors, and enhancement of dopaminergic and noradrenergic activity. These differential effects have been linked to the development of psychotic behaviour. The facilitation of dopaminergic function by buspirone may be more clinically significant than hitherto thought and is possibly the mechanism through which this drug induces mania.

In this case, it could be argued that the onset of mania had nothing to do with buspirone. However, this appears unlikely: the patient had been withdrawn a number of times from alcohol and had taken disulfiram in the past, both without adverse effects. There have been no reported interactions between buspirone and disulfiram. The temporal relationship of being on buspirone and becoming manic, would seem to preclude any role that alcohol may have had in the onset of his symptoms.

If buspirone does cause hypomania, its use in alcoholics, or drug addicts, although attractive, should be carefully monitored and the relative risk of precipitating a psychotic illness weighed against the attraction of using an anxiolytic which is reputed to be free of abuse or dependency.

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Educating the psychiatrist of the 21st century

SIR: As I read Cawley's Lecture (*Journal*, August 1990, **157**, 174–181), with its persuasive *tour d'horizon*, I found myself becoming increasingly restive. Finally, and doubtless because I live abroad, I spotted the trouble – his title was wrong. It should have been "Educating the British NHS consultant psychiatrist in the 21st century". If we hold psychiatry to be a discipline like others that transcends local administrations and national frontiers, the education of psychiatrists in general is another matter.

As psychiatrists go, the British consultant is a peculiar animal in ways that I can only outline here. He or she is employed in large hospitals and institutions, and would be classified as a hospital or a government psychiatrist in some other countries. He has concerns with 'management', and now 'audit', which are of no interest or incomprehensible to many psychiatrists elsewhere. He is one of a team of public employees that deals with the patient, and he does relatively little clinical work by himself, and even less of the kind sustained for months or years with individual cases, from their start to their finish. He may go into the outside world with his team but he does not belong to it, and therefore has scant familiarity with the main mass of psychiatric disorders, even though they are often as destructive to human welfare as the atypical fraction he meets in hospital. Indeed, as Professor Cawley puts it, he and his team "may have little direct experience of the vast majority of the clinical problems in the field in which they claim expertise".

Far from taking psychoanalysis near its centenary peacefully for granted as integral to the study of mind, culture, and society, the British consultant psychiatrist sees it still as totally controversial, if not crackpot, and of no everyday clinical relevance, while quite unaware that there are neighbouring countries (e.g. Germany) where psychoanalytic therapy is funded by the health service, widely available, and reinforced by university departments. Although seldom possessing serious knowledge of it, he can, as Professor Cawley sadly observes, be ferocious in belittling psychoanalysis, unless he belongs to a small eccentric minority who have a habit of forsaking psychiatry altogether for psychoanalytic practice amid a non-medical fraternity almost confined to London.

If we forget, as I fear Professor Cawley has done, that this animal is only one type of psychiatrist, but by no means the world over the common one, we can easily agree that it is absolutely essential for the psychiatrist in training to be schooled in "modern management techniques", in collaborative research with neuroscientists, epidemiologists, and others, and in sharing his patients with a multiform team, all of which could seem esoteric luxuries and bizarre priorities elsewhere. Also, we can then easily forget to ask the vital question whether this is the psychiatric animal that we want to go on producing at all.

However, once the question is asked, we are up against it because we seem to have no choice but to go on producing what we do. Professor Cawley looks for innovations in psychiatric training in Britain but he does not examine the obstacles to effecting any very substantial changes, let alone fundamental ones. As I have pointed out (Bourne, 1988), these obstacles are the more huge for being invisible because they are buried in the embryology of the British Psychiatry – not only in his basic medical education, in his prejudicial studies, and in his selection as a medical student in the first place, but in his schooling from early adolescence onwards, and in the national character in which he is bred, and the society and its administrative and political styles and structures that he takes for granted.

Yet the question surely should be asked so that we proceed by choice rather than by habit, and also because asking questions and making choices is the first step towards overcoming the hugest obstacles.

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CORRIGENDUM

Journal, November 1990, **157**, 778 (Adityanjee). The name should read "Adityanjee" only and not "Adityanjee, P. Das". The first reference should read "Adityanjee (1987)" and the second reference should read "Adityanjee, Das, P. & Chawla, H. M. (1989)".

A HUNDRED YEARS AGO

Harvelian Society. General Paralysis of the Insane

At a meeting held on March 20th, Dr Savage read a paper on General Paralysis of the Insane, which he considered to be a disease of higher civilisation. It was rare in the highlands and wilds of Great Britain and Ireland, and also in the negro of the Southern States. Dr Savage looked upon general paralysis as a premature decay frequently having a local origin. In earlier symptoms of this disease extreme difficulty was often experienced in distinguishing between causes and early symptoms, for in many cases the causes of general paralysis might often form signs of the disease. As a result of extensive observation, Dr Savage finds it more commonly met with in middle-aged married men, inhabitants of cities, flesh eaters, and drinkers of alcohol. When the onset is gradual, the finer social and muscular adaptations fail, and weakness in mind and body gradually becomes apparent. In cases which occur suddenly, the storm may assume the character of a convulsive seizure, or an attack of emotional excitement, or one of mania. Ataxy also may be the first symptom of general paralysis. In this form there will generally be muscular defects of the hands and tongue out of proportion to the progress of the ataxy, and the symptoms will be either those of exaltation or of hypochondriacal melancholia; whereas, if the insanity be that of the ordinary ataxy, it will be of the suspicious and "persecuted" type. Temporary aphasia is one of the most striking warning symptoms of this disease. In such cases the patient, without any real cause of after slight excitement or fatigue, becomes aphasic. The attacks may recur, and this aphasia is usually marked, before changes in the handwriting become apparent, though this symptom is an early one in cases of general paralysis. Then facial expression is very early affected. Syphilis plays so important a part in general paralysis that Dr Savage says: "If after a history of syphilitic cranial nerve lesion there are any signs of nervous instability, there is real reason to fear that general paralysis may be the result. Any local cranial lesions, especially such as depend upon syphilis, may originate the degeneration of general paralysis". Dr Savage observes from his notes that neuralgia, headache, sciatica, rheumatic pains, and the like, were almost universally recorded as having been noticed a year or more before general paralysis was recognised. Double sciatica is not a symptom to be overlooked, and if it is recurrent and associated with any change in habits or character, it is a warning not to be disregarded. Among the warnings of a "mental" nature, the gradual loss of power of social accommodation is one of the earliest and most marked. Memory for recent events becomes defective; loss of the power of attention and a want of persistence are very well marked. In early general paralysis stupid stealing and thoughtless indecency are the common forms of its manifestation. The early "general paralytic" easily becomes drunk, and is easily poisoned. Changes of temper and character are probably the most common of all the changes which are noticed in early general paralysis. Alternations of buoyancy and depression are of bad import. The sudden outbreak of mania is a frequent precursor of general paralysis, and this specially interesting when it assumes the form of delirious mania. Warnings of general paralysis may be present for years, and almost certainly they are present for a year at least before the symptoms induce even the sensitive specialist to scent out the disease.

Reference

Lancet, 5 April 1890, 753.

Researched by Henry Rollin, Emeritus Consultant Psychiatrist, Horton Hospital, Surrey.

British Journal of Psychiatry (1991), **158**, 139–141

Books Reconsidered

On Becoming a Person: A Therapist's View of Psychotherapy: Carl Ransom Rogers*

Carl Rogers, who died in California in 1987 at the age of 85, was ranked in a late-1970s survey of American clinical psychologists and counsellors as being the psychotherapist who most influenced them, surpassing such stalwarts as Sigmund Freud and Joseph Wolpe. This book was the fourth one that he published and is reputedly the most renowned. In the UK it is his best-selling book, being reprinted on average every 18 months and having sold about 4000 copies in the last two years.

This book consists of 21 papers written between 1951 and 1961 while Rogers was responsible for first the University of Chicago Counselling Centre and subsequently the Psychotherapy Research Section of the Wisconsin Psychiatric Institute, which he joined in 1957. Many of the papers were presented to general audiences which Rogers had been invited to address, and all are written in a non-technical style which is easily understood. While most of the contributions had already been published elsewhere, they were generally not readily available to those who wanted to follow Roger's thinking more closely. Gathering the papers together in one place meant that his ideas could reach a wider readership.

Incidentally, a more accurate subtitle for the book would have simply been *A Therapist's View,* since some of the later papers in this volume are concerned with issues which go beyond psychotherapy to include education, creativity, the growth and use of knowledge in the behavioural sciences, and intergroup tension. Indeed, one of these papers was contributed not by Rogers but by an academic educationalist who attended one of his short courses and who wrote an appreciative letter on his reactions to this immediately after the course as well as a year later. The majority of the papers, however, are concerned with psychotherapy and with what it means to be psychologically healthy.

There are three basic assumptions which seem to run through these papers and through his writings generally, and which may be used to illustrate his ideas. First and foremost is his optimistic belief that people will live a constructive and fulfilling life provided they are fully aware of their own feelings and experiences and use this knowledge to guide their behaviour. This point of view is most clearly and convincingly argued in chapter nine, entitled "A therapist's view of the good life: the fully functioning person". In this paper he suggests that if we are alert to all our feelings, then the expression of negative acts will be tempered by our awareness of positive experiences. In addition, being fully sensitive to our feelings will provide us with greater information on the most appropriate course of action for us to take and will also give us more feedback on where we may have gone wrong.

Rogers' own therapeutic way of working, which he did not recognise constituted a new, personal and distinctive approach to therapy until 1940, is described in the first chapter of the book in an autobiographical piece, unpretentiously entitled "This is me". It grew from his realisation that the diagnostic, psychoanalytic and rather mechanistic approach that he had at first adopted in the treatment of delinquent and underprivileged children at the Rochester Guidance Centre, where he initially worked, was not always successful. He somewhat cryptically recounts in this chapter one incident in particular which led him to question the value of this approach and to develop a more satisfying way of working.

During the course of many interviews with an intelligent mother, he had been sensitively trying to point out to her, but with little success, that the unruly behaviour of her child was due to her early rejection of the boy. After they had both agreed that, since they did'not seem to be making any progress, treatment should be terminated, she then asked Rogers whether he counselled adults. Since he did, she asked for help and began to talk about the unsatisfactory nature of her relationship with her husband, and her own sense of failure and confusion. Although Rogers does not explicitly state that the boy's behaviour improved, he does write that therapy was ultimately successful.

Such events as these gradually convinced him that it was the patient or client who knew what the problems were and that the role of the therapist should be to provide a conducive atmosphere in which clients could direct their own therapy. He came to call this approach client-centred therapy. Later the word 'client' tended to be substituted with that of 'person' to emphasise that his ideas were not confined to what constituted successful therapy but could also be applied more generally to facilitate constructive behaviour in other spheres of life, as illustrated in the later chapters of this book. Although not happy with the term 'client', he preferred it to that of 'patient' which had adverse connotations of dependence and passivity.

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*1961. London: Constable. £6.95

One notable implication of this philosophy for the training of therapists, which is not always readily appreciated or understood, is that his style of working is not necessarily the approach which should be adopted by every clinician. In fact, therapists should be encouraged to develop their own therapeutic outlook based on their own experience of helping others. Consequently, Rogers was opposed to setting up organisations which would teach his own mode of working, since this would contradict his fundamental belief that we should trust and be guided by our own experiences. It is worth stressing, however, that it does not necessarily follow from client-centred theory that such organisations would subvert rather than propagate this central teaching. Despite Roger's scepticism, a number of national and international associations concerned with the development of the client-centred approach have been running for some years, although it is not yet clear whether his forebodings have been confimed.

Inextricably related to Rogers' belief in the potentially positive nature of human beings is his view that this aspect will only predominate and flourish if a person's feelings are genuinely understood and accepted, even if they are not allowed to be acted on, should they be seen as being socially destructive. Rogers thought that the acknowledgement and acceptance of such harmful urges should help to defuse them. It is somewhat surprising then that Rogers did not include in this collection his paper entitled "The necessary and sufficient conditions of therapeutic personality change" (*Journal of Consulting Psychology*, 1957, **21**, 95–103), in which he succinctly laid down the three therapeutic attitudes that were necessary and sufficient for therapeutic improvement to occur. Although the theoretical position in this paper is described in chapter 14, "Significant learning: in therapy and in education", the original exposition would have provided a useful summary for the lay reader and would have considerably enhanced the value of this collection.

The most concise statement of Rogers' view of the process of therapy is to be found in a paper called "The process equation of therapy", which, although published at the same time as this book in the *American Journal of Psychotherapy* (1961, **15**, 27–45), was also unfortunately not included in it. In this paper, he proposes that this equation can be crudely and tentatively expressed as: "The more the client perceives the therapist as real or genuine, as empathic, as having an unconditional regard for him (sic), the more the client will move away from a static, unfeeling, fixed, impersonal type of functioning and the more he will move toward a way of functioning which is marked by a fluid, changing, acceptant experiencing of differentiated personal feelings." (p. 40). This neatly captures the three essential therapeutic or core conditions of genuineness, empathy, and unconditional positive regard, as well as the kind of changes that can be observed taking place in the client. A more detailed explication of these changes, which were based on trying to listen as naively as possible to many hours of recorded therapy sessions, can also be found in chapter seven entitled "A process conception of psychotherapy."

The third main assumption underpinning Rogers' approach is his belief in the importance of endeavouring to empirically test ideas. In chapter 10, "Persons or science? A philosophical question", he reconciles what he previously thought was the incompatible nature of objective science and his highly subjective approach to therapy by recognising the essentially personal character of the creative process of scientific thinking. When first studying an unfamiliar phenomenon such as psychotherapy, he believed it necessary for scientists to immerse themselves in their relevant experience, from which may emerge tentative hypotheses concerning its structure and the underlying processes which determine it. Consequently, in his attempt to understand and objectify the process of psychotherapy, Rogers was the first therapist to systematically record electrically what took place. The earliest verbatim account of a complete case of therapy ever published is that of Bryan in Roger's *Counselling and Psychotherapy* (1942, Boston: Houghton Mifflin). His theory of therapy was based on listening to and trying to understand cases such as these.

Client-centred theory generated a considerable amount of research up until the mid-1970s, much of which was carried out by Rogers and his associates. A highly commendable characteristic of this work was its attempt to examine the client-centred theory of the relationship between the process and outcome of therapy, a feature which current psychotherapy research would do well to emulate. Enticing but largely uncritical snippets of this imaginative research are presented in chapters 11 and 12, respectively entitled "Personality changes in psychotherapy" and "Client-centered therapy in the context of research". The most detailed review of research on the therapeutic effectiveness of the three core conditions as viewed from a strictly client-centred perspective is, however, to be found elsewhere in Gurman's chapter "The patient's perception of the therapeutic relationship" in *Effective Psychotherapy* edited by himself and Razin (1977, pp. 503–543, New York: Pergamon).

Perhaps the less than positive results of later studies on the therapeutic role of these therapist's attitudes and the decreasing youthfulness of the original workers led to the decline of research in this area. However, more recently there has been a resurgence of empirical interest in the role of the therapist–client relationship by both behaviour therapists and empirically-minded psychodynamic clinicians, and which hopefully may help to resolve the inclusive nature of previous research. Although Rogers drew attention to this point in his earlier book *Client-Centered Therapy* (1951, Boston: Houghton Mifflin), there is a surprising dearth of research by client-centred workers on the therapeutic process as seen by the client. If clients know what the problems are initially, then presumably they are also in a better position to realise how these problems were overcome. It may be unrealistic, however, to expect people who are psychologically distressed to be able, or to be particularly interested in trying, to understand in detail their own therapeutic process. Nonetheless, clients' reflections on their own treatment may provide useful leads in furthering our knowledge of psychotherapy.

While *On Becoming a Person* may serve as an overview of the client-centred theory of human behaviour, people who are principally interested in how Rogers actually carried out therapy, particularly as reflected in the verbal exchanges which took place between him and his clients in recorded sessions, would do better to turn to his two previous volumes *Counselling and Psychotherapy* and *Client-Centered Therapy*, which contain many examples of such interchanges with indispensable annotated comments. Although Rogers claimed to have been more directive in those earlier days, there are fewer published verbatim illustrations of his later less directive approach. One highly commendable exception is the filmed half-hour demonstration session with Gloria, which has been fully transcribed in Rogers' chapter in Burton's *Operational Theories of Personality* (1974, New York: Brunner/Mazel). However, it should be borne in mind that at present there is little evidence to suggest that the client's increasing self-awareness of feeling, which Rogers focuses on as being indicative of and necessary for therapeutic improvement, is related to outcome, let alone a determinant of it.

Hopefully, the more popular appeal of *On Becoming a Person* will continued to promulgate Rogers' belief in the trinity of subjective experience, objective scrutiny and the essentially positive nature of humanity in helping us, not only to further understand the process of psychotherapy, but also to lead more fulfilling lives.

Duncan Cramer, *Lecturer in Social Psychology, Department of Social Sciences, Loughborough University of Technology, Loughborough*

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Book Reviews

Editor: Sidney Crown

Psychiatry Around the Globe: A Transcultural View (2nd edn). By Julian Leff. London: Gaskell. 1989. 226 pp. £10.00.

This is a remarkable book. The first edition was published in 1981 when transcultural psychiatry was being strongly influenced by sociopolitical concerns. The second edition has reached our desks when the subject has become yet more popular and is now a training requirement for many health professionals. To use Murphy's phrase – transcultural psychiatry in the UK has 'come of age'. The second edition of this book, therefore, is both a result and cause of this important development. Tutors, consultants, Mental Health Act commissioners, MRCPsych candidates and librarians are recommended to possess it. Similar and appropriate praise is given in the foreword by Kleinman.

> "Leff clarifies which questions have been answered, which problems remain to be solved and which have been refined and restated .. This fine volume is popularisation in the best sense. I believe it deserves and will receive a very wide readership .."

This reviewer underlines what Kleinman has said. Leff has indeed also shown his stature by listening to his critics and then modifying the chapters which described the schema for development of emotional differentiation, so that they are less culture-bound.

There could yet be a need for a further edition (or a new book) to enlarge further on transcultural aspects of psychotherapy, to provide examples of teaching curricula, give an account of qualitative research methods and to describe cultural aspects of psychopharmacology. Let us hope that Leff with his enviable writing talent and academic resource will address other transcultural topics, and that Gaskell Press will retain their interest in these vital aspects of psychiatric practice.

John Cox, *Professor of Psychiatry, University of Keele*

Understanding Economic Behaviour. Edited by Klaus G. Grunert and Folke Olander. Dordrecht: Martinus Nijhoff. 1989. 440 pp. £57.00.

The more that non-economists discover about economics the more surprised they become. This is not (usually) because the subject produces surprising but true results but because economists, particularly in the West, seem to adopt such strange assumptions about human behaviour. Psychologists, sociologists and social anthropologists are especially likely to be critical, if they do not dismiss the subject completely.

Economists (who are not usually noted for their modesty) can generally live with this scepticism but it is desirable that at least some members of the profession should be open-minded enough to hear what other students of human behaviour have to say and should also attempt to explain why the subject has developed in the way it has. This book provides an excellent example of such a dialogue. It is based on a conference held by the International Association for Research in Economic Psychology in Denmark in 1987 and includes papers by mainstream economists as well as by specialists in economic psychology.

It may be worth sketching the traditional approach of Western economics, which is the subject of critical (although not necessarily hostile) review in this volume. There are two strands. The first, which was set out most notably in Lord Robbins' book *The Nature and Significance of Economic Science*, argued that economics is an axiomatic system based on self-evident truths about human behaviour. Since the initial assumptions were true, all statements derived logically from them must also be true. The second strand was described in Professor Milton Friedman's book *Essays in Positive Economics*. Friedman stated that the success of economic theories should be judged by their predictive power. The truth of the underlying hypotheses was unimportant in itself. Linking the two strands is the notion of the individual acting rationally to maximise his utility – an idea which at its best is a powerful organising concept but which at its worst is simply tautological (since all actions must be rational in respect of some objective).

Economic psychology is concerned with individual behaviour and particularly emphasises the relation between actions and *perceptions*. The main research method consists of empirical research, which includes laboratory experimentation (a technique which most economists would regard as impossible). The papers in this volume are divided into five groups. The first is concerned with economic factors and well being. They ask, for example, whether any relationship can be discovered between the economic prosperity of a nation and the happiness of its inhabitants. The second group is concerned with perceptions of macro-economic phenomena (e.g. inflation and unemployment). The third considers details of individual decision-making. The fourth group examine consumer behaviour, and the fifth group raises more general questions about economic analysis. It includes an account of animal experiments which seek to discover demand equations from the behaviour of rats and rhesus monkeys.

The editors provide a useful introduction. Psychologists interested in economics and economists interested in psychology will learn something from all the papers.

ALAN BUDD, *Visiting Professor of Economics, London Business School*

Festina Lente: A Psychiatric Odyssey. By HENRY R. ROLLIN. London British Medical Journal/The Memoir Club. 1990. 194 pp. £14.95.

For half a century, Dr Rollin has participated in some of the most significant developments in British psychiatry. It would be difficult to find a more entertaining guide to the discipline's recent past than Rollin, whose recollections are written in a conversational and engaging style, which combines both humour and perceptive observation. While the events of 20th-century psychiatry are reasonably well known, the intriguing aspect of this book, especially to younger generations, is Rollin's evocation of the general day-to-day atmosphere of the previous era. He captures well the rigid hierarchy of the 'total institution' of pre-war days, where the superintendent reigned supreme, and where the junior medical officers were required to live-in and to ask for permission to leave the asylum overnight.

The advent of insulin coma therapy, pre-frontal leucotomy and electroconvulsive therapy – 'the high noon of physical methods' – is recounted and Rollin vividly conveys the excitement of the times and the near messianic faith in the new therapies. He is honest enough to describe his own temporary 'conversion' but also his subsequent disappointment at the failure of physical treatments. With the exception of ECT, Rollin's judgement of this period is severe, with particular condemnation being reserved for psychosurgery.

Of great contemporary relevance is Rollin's account of the first moves, in the late 1950s, to close mental hospitals. The now familiar issue of hospital versus 'community' care was being fiercely debated, and Rollin sounded a largely unheeded warning about the dangers of emptying asylums in the absence of an adequate alternative:

> "[many patients] will join the army of vagrants and elbow each other off the park benches, or lengthen the queues outside the doss-houses. Others still will embarrass the courts and prisons where their crimes, usually petty and purposeless, unless interpreted as a plea for care and protection, have landed them."

Many other aspects of the cultural history of psychiatry in the last 50 years are reflected in the book. the changing presentation of mental illness; the rise and fall of psychoanalysis; the fate of the mentally abnormal offender; and the development of out-patient clinics.

Of course, Rollin does not confine himself merely to the medical world and his lifelong interest in the Arts is given generous space, with affectionate memories of the theatre in Leeds and London; jazz music in New York, and classical music at Oxford. Attention is also given to his appraisal of Shakespeare, Byron, Shaw and Joyce As he concludes:

> "Psychiatry .. spills over into so many other disciplines that it affords a golden opportunity for the dilettante – which is essentially what I am – to flourish."

Rollin's wide cultural interests have surely increased rather than diminished his ability as a psychiatrist and it would be sad if such 'dilettantes' did not continue to find a place in contemporary psychiatry.

ALLAN BEVERIDGE, *Consultant Psychiatrist, West Fife District General Hospital, Dunfermline, Fife, Scotland*

Dysthymic Disorder. Edited by S. W. BURTON and H. S. AKISKAL. London· Gaskell 1990. 130 pp. £7.50.

This well produced paperback contains the proceedings of the first symposium devoted entirely to discussion of the new DSM–III–R concept of dysthymic disorder. In his foreword, the late Professor Max Hamilton points out that 'dysthymia' covers a wide variety of conditions and under one name or another has always been with us. Perhaps the best known traditional term has been 'chronic neurotic depression' DSM–III–R dysthymia requires low grade chronicity of depressive symptoms of more than two years, early and insidious onset, a persistent or intermittent course, concurrent character pathology and, finally, is an "ambulant disorder compatible with stable social functioning". The North American influence behind the inclusion of dysthymia in DSM–III and DSM–III–R owes much to the work of Professor Akiskal of Tennessee, who is co-editor of the book and one of the major contributors. From his work on mild chronic depressives, Akiskal has derived a complex classification and subclassification of dysthymics. This is drawn largely it seems from US private practice experience. Subcategories include subaffective dysthymia with shortened latency of rapid eye movement on electroencephalography and good response to tricyclics, and also 'character spectrum disorder' reflecting a *melange* of dependent, histrionic and antisocial traits

From the text of Akiskal's opening chapter it is clear that a major US concern has been to salvage a mild, chronic subgroup of depressives who heretofore were dismissed as hopelessly character disordered and suitable only for psychotherapy or to be dismissed as beyond help. All this is admirable, but, as papers by Tyrer and by Goldberg from the UK demonstrate, the scientific validity of DSM–III–R dysthymia is difficult to substantiate. Sievewright & Tyrer report their failure to confirm any of the distinguishing features of dysthymia except for chronicity, in a prospective study over one year of a small cohort of patients who initially satisfied diagnostic criteria. In particular, the diagnosis proved unstable, most patients developing other affective disorders over time, especially anxiety disorders. Another UK study by Murphy & Checkley identified a group of 24 dysthymics among 117 depressives who were first attenders at the Maudsley emergency clinic over three months. Compared with major depressives, dysthymics were distinguished by earlier onset of symptoms, a higher anxiety scale score and more frequent early separation. Multivariate analysis failed to substantiate Akiskal's separation of subaffectives from character spectrum disorder. These authors conclude that the dysthymic–major depression dichotomy is valid on clinical grounds but they did not address a further concept of 'double depression' when a dysthymic experiences a major depressive episode.

Goldberg & Bridges, drawing on their by now extensive studies of general practice morbidity, declared that "dysthymic disorder is a new plastic box for some rather old wine". They echo the by now traditional British distrust of US classificatory innovation. In discussion, Goldberg articulates concern that premature acceptance of dysthymia as a diagnostic concept may lead to inappropriate and unnecessary prescribing of drugs in place of what he would consider more supportive help in the shape of counselling and social work. There are other contributions on biochemical aspects, personality and epidemiology, none of which sheds further light except to emphasise the heterogeneity underlying the dysthymia concept. Working clinicians will be forgiven for feeling confused and disappointed that little research evidence is yet available to support a scientifically based taxonomy at the milder end of the affective disorder spectrum. Trainees will scratch their heads after reading this book and wonder what kind of answer examiners will expect to questions about classification. Examiners on both sides of the Atlantic should reflect on the ethics of even daring to set a question on this subject in the present state of uncertainty. For a succinct summary of what is now known, the book is worth looking at.

J. Fahy, *Department of Psychiatry, Regional Hospital, University College Galway*

The Control of the Hypothalamo-Pituitary Adrenocortical Axis. Edited by F. Clifford Rose. Connecticut: International Universities Press. 1989. 446 pp. $65.00.

This book is based on a symposium held in London in early 1988. The meeting was planned by Professor Mortyn Jones who sadly died a few weeks before it took place. The volume is dedicated to his memory and there is a tribute to him by the editor and two of Professor Jones' colleagues. There is much in the book that he would have enjoyed. The authors are acknowledged experts in their field and the book spans both basic and clinical research with several chapters bridging the gap. Throughout there is an emphasis on both the importance and the limitations of experimental techniques. It is fascinating to see how newer technologies (many pioneered by Professor Jones) have allowed insights into the physiology of the hypothalamus. The 'brain' and adrenal ends of the axis are not forgotten (as sometimes happens in such volumes) and there is an emphasis on the integration of the whole system. Many chapters have sections on outstanding problems in the topic which are particularly useful and in keeping with the high scientific quality of the book as a whole.

Inevitably there are the drawbacks related to symposium proceedings, with some repetition and inconsistency. The book is not for the faint hearted and probably most suited for potential specialists. Nevertheless, no psychiatrist should measure cortisol in their patients without having at least glanced at a book such as this – indeed, preferably, this one.

Nicol Ferrier, *Professor, MRC Neurochemical Pathology Unit, Newcastle upon Tyne*

Dementia Disorders: Advances and Prospects. Edited by C. L. E. Katona. London: Chapman & Hall. 1989. 236 pp. £30.00.

Katona has edited a compact but wide-ranging book which demonstrates the plurality of dementia disorders and clearly discusses both neurodetail and service. For a multi-authored book covering some complex topics the writing is consistently clear and accessible, and the very detailed contents list with multiple subheadings throughout the book makes for easy reference.

Ineichen begins by reviewing epidemiological evidence and encouragingly suggests that the figures of 10% of those aged over 65 years and 20% of those aged over 80 years are a considerable overestimate of dementia; however, he also says that the increase in numbers of very old people and increase in survival rate of dementia sufferers may be severely underestimated. Tym, in a full chapter on diagnostic assessment, ends with a plea for continuing to refine assessment procedures for diagnosing early stages of dementia. No future physiological measure can substitute for overall clinical appraisal of the demented patient.

Chapters follow on molecular neuropathology, neuropharmacology and multi-infarct dementia. Friedenberg *et al* present an excellent chapter on subcortical dementia, likewise Philpot & Burns on reversible dementias. In the chapter on AIDS and dementia, one of

Katona's conclusions is that we need to know more of the natural history of the disorder to plan future provision. This is particularly important for younger patients with mild to moderate cognitive impairment but relatively little physical illness, whose care needs we are unclear about at present.

Volans gives a comprehensive overview of assessment from a psychological perspective but offers little on management. This is surprising as it is reiterated in other chapters that we still seem to be a long way off physiological treatment and thus psychosocial management is our only avenue. Norman's contribution on models of care and defining adequate standards is characteristically excellent. Lindesay, in a wide-ranging chapter on future models of care in changing times, takes us via robotic home helps and talking pill-boxes to the inevitable conclusion that, whatever the model, the quality of care depends on staff "recruitment, numbers, training, management, support and morale".

This is a very good book both for trainees and for use in refreshing those engaged in running a service.

JANE GARNER, *Consultant Psychiatrist, Department of Old Age Psychiatry, Chase Farm Hospital, The Ridgeway, Enfield EN2 8JL*

Yearbook of Psychiatry and Applied Mental Health 1989. Edited by J. A. TALBOT, A. FRANCES, D. X. FREEDMAN, H. Y. MELTZER, J. E. SCHOWALTER and HERBERT WEINER. London: Wolff Medical Publications. 1989. 479 pp. £40.00.

Yearbook Medical Publishers Incorporated survey nearly 700 US and overseas medical and allied health journals from which are abstracted articles for their series covering clinical topics from anaesthesia to vascular surgery and, in this case, psychiatry and applied mental health. The most recent articles reviewed were published in June 1988. Abstracts from these articles are presented under 13 broad headings including "Biological psychiatry", "Genetics", "General clinical topics", "Clinical psychiatry", "Psychotherapy", "Psychopharmacology, law and psychiatry", and "Community psychiatry".

The abstracts are clearly written and each is followed by one or two sentences of comment from an editor. The result is a fairly small volume which is an excellent reference for quite recent research on any topic. The index (covering subjects and authors) is competently arranged. Many of the articles are in journals not usually read by the average psychiatrist, particularly on this side of the Atlantic. Nevertheless, articles are abstracted from the major European journals, including this Journal, of course!

A random sampling of topics showed that 13 articles were included on antidepressants and post-traumatic stress disorder while there were six on involuntary commitment. The latter were abstracted from North American journals but European readers will be interested in the discussion of the conflict between civil liberties and the need for involuntary treatment.

In the section on psychotherapy, cognitive and behavioural approaches predominate, reflecting the diminution of psychoanalytic approaches in the practice of American psychiatry.

Overall, this book could be recommended as an excellent reference, less thorough by far than the *Index Medicus*, but easier and quicker to use.

R. J. DALY, *Professor of Psychiatry, Cork Regional Hospital, Cork*

Fundamentals of Monitoring Psychoactive Drug Therapy. By C. LINDSAY DEVANE. Baltimore: Williams & Wilkins. 1990. 288 pp. £23.50.

The concept behind this book is excellent. It sets out to provide basic information on the clinical pharmacology of the drugs used in everyday psychiatric practice. This has become a much neglected area, often overshadowed by the more exciting, although unresolved, developments on mechanisms of action and neurochemical pathologies of psychiatric disorders.

The opening chapter provides a review of the history of psychotropic drugs followed by a rather brief section on neurotransmitter function. This is followed by excellent, concise sections on the basic principles of pharmacodynamics and pharmacokinetics. These provide adequate knowledge for the clinical psychiatrist and are readily understandable The main omission is the lack of more than a passing mention of the 'first pass' effect A final clinical section, intended for non-clinicians, is superfluous to psychiatrists.

The subsequent chapters review the drugs used for treatment of particular types of disorder mood disorders, the psychoses, anxiety and insomnia, childhood mental disorders and substance abuse. Each chapter follows a standard pattern. An initial overview of treatment is followed by a detailed 'database' of 6–12 pages each on specific drugs used as examples within a particular class (e.g. for mood disorder, lithium, imipramine, phenelzine and trazodone are covered). This is then followed by a brief, largely tabular comparison of all drugs within particular classes. Finally, each chapter has a section of short paragraphs on essential data and differences relating to other commonly used drugs not mentioned in the detailed 'databases'. For example, amitriptyline, clomipramine, fluoxetine and others are included under the heading of 'cyclic antidepressants'. This overall format avoids duplication of information and provides a fairly readable book.

There are omissions, some of which probably reflect the book's American origin and therefore the fact that some drugs have only recently or not yet become available there. More information on the newer 5-hydroxytryptamine (5-HT) specific drugs could be provided as well as on lofepramine. In addition, data on relative risk in overdose might be of value. In the "Psychoses" chapter sulpiride is not mentioned, nor is there a detailed 'database' for any depot preparation. In chapter four on "Anxiety and insomnia" propranolol and barbiturates should perhaps be awarded more space and in chapter six on 'Substance abuse" the pharmacology of alcohol itself is not covered. Also missing is a chapter on epilepsy which would be of value to most psychiatrists.

Overall, then, a useful book for the shelf of the consultant and helpful for the trainee. While the subject matter may not be immediately attractive, it is important, and if the psychiatrist on the mental health team does not understand drug pharmacology, who is going to? This book provides a reasonably easy access to the necessary information. Unfortunately it is a little spoilt by some of the omissions, but aside from these it seems fairly complete.

STEPHEN J. COOPER, *Senior Lecturer, Department of Mental Health, The Queen's University of Belfast*

Clinical Pharmacology in Psychiatry from Molecular Studies to Clinical Reality. Edited by S. G. DHAL and L. F. GRAM. 1989. 330 pp. DM98.00.

This volume is a collection of papers presented at an international meeting held in Norway in 1988. As such the chapters are very heterogeneous. Although of a high quality, the immediate relevance to clinical psychiatry is not great. The subject matter ranges from the classification of receptors, through the genetics of drug metabolism, to drug level monitoring.

There is a useful chapter on drugs affecting D1 and D2 receptors; evidence is now emerging that D1 receptor blockade is important in the action of some drugs such as clozapine and the thioxanthines.

A chapter on meta-analysis suggests that serotonin-specific reuptake blockers are less effective as antidepressants than some older tricyclics.

Several chapters are concerned with individual variations in drug metabolism. In particular, hydroxylation measured by debrisoquine metabolism is deficient in 5% to 10% of Caucasians; these individuals develop higher than expected blood levels of many tricyclic antidepressants and some phenothiazines, with consequent side-effects and intolerance. Some antipsychotic drugs and some antidepressants inhibit the hydroxylation enzymes, resulting in pharmacokinetic interactions between these drugs. Tissue from human liver banks can now be used to assess drug metabolism as part of the development of new drugs, without exposing living subjects to the drug; this is surely an important advance.

The book is recommended for psychiatrists wishing to know more about the metabolism of psychotropic drugs.

JOHN COOKSON, *Consultant and Honorary Senior Lecturer, The Royal London Hospital, London*

Design and Analysis of Reliability Studies. The Statistical Evaluation of Measurement Errors. By G. DUNN. Oxford: Edward Arnold. 1989. 198 pp. £25.00.

This excellent book deserves a place on the bookshelf of anyone concerned with the statistical problems of assessing the dependability or reliability of measurements. Starting from the assumption that the reader has a "level of statistical competence ... covered by most elementary statistics courses", the book is neatly divided into three self-contained sections. In the first section, three chapters cover the basic types of mathematical measurement models (including binomial and Poisson processes as well as normal distributions), coefficients of agreement/disagreement and indices of reliability, consistency and stability. The second section is a single chapter on the often neglected topic of experimental designs for reliability studies (including consideration of sample size estimation). The final and longest section consists of three chapters on inferential methods used in the analysis of data from reliability studies; two chapters concentrate on methods appropriate for interval data while the third deals with categorical and ordinal measurements. Some essential mathematical concepts are described in detail in a series of appendices, and a list of major statistical computer packages which include the techniques dealt with in the book is also provided.

The techniques covered constitute a comprehensive guide to the statistical methods widely used in reliability studies. All of the methods are explained clearly, supplementing the rigorous mathematical descriptions supplied. The liberal use of worked examples clearly drawn from real clinical situations are particularly helpful. The mathematical notation used follows standard conventions and is consistent throughout the book.

The book contains one glaring omission. In their seminal paper, Bland & Altman (1985; *Lancet, i*, 307–308) argue forcibly that the assessment of reliability is not entirely a matter of statistical significance testing. Indeed, they show an example in which one of the statistical techniques advocated in this book leads to an erroneous conclusion. Bland & Altman advocate a confidence limit approach to reliability estimation, which essentially supplies an estimate of the probability that the difference between two supposedly identical measurements will lie within a clinically acceptable range. This approach usefully combines both statistical and clinical significance, but is totally ignored by Dunn.

There is also some doubt in my mind that the author has correctly identified his potential audience. A level of mathematical expertise up to and including a working knowledge of matrix algebra is required, and I am not convinced that many non-statisticians will possess that. Reasonably numerate researchers looking for a 'cookbook' of suitable methods should not be daunted by the complexity of the formulae presented, however; this is a book which will richly reward perseverance with any unfamiliar mathematical concepts.

E. B. FARAGHER, *Department of Medical Statistics, University Hospital of South Manchester*

AIDS, Drugs and Prostitution. Edited by MARTIN PLANT. London: Routledge. 1990. 213 pp. £30 00.

This book consists of a collection of papers, some heavily referenced others not, describing research into prostitution, and originates from a workshop held in Edinburgh in February 1989 It consists of an introduction and conclusion by the editor and 10 chapters by 14 authors with over 40 tables, although some of the data has already appeared in journals. The major themes explored are the reasons for prostitution, the use of condoms in working and non-working relationships, the amount of drug use by prostitutes, and the fact that except in Africa, to date the majority of HIV infection among prostitutes is related to injection drug use rather than heterosexual spread. Many of the chapters have descriptive introductions concerning the history and manner of prostitution in various parts of the world.

While posing the question how have prostitutes and clients responded to AIDS, the book actually only deals with prostitutes and their reports of clients' views or behaviour. No actual data on clients is presented which seems surprising since presumably if one can recruit prostitutes from sexually transmitted disease clinics one can recruit clients. There are some common problems with all of the studies presented which are best exemplified by Dr Darrows' review of seroprevalence among prostitutes in the US. The majority of reports have less than 100 participants, some as few as 10, yet we are constantly told that prostitution is widespread. While there are numerous varieties of prostitutes from street walkers to those in massage parlours and escort agencies it is worrying that the majority of work and the conclusions reached are based on street prostitutes.

In the concluding chapter the editor suggests that the different methods employed by the various authors, together with the uniformity of their results, strengthens the validity of the data. In fact all the studies used only two methods of recruitment, either via sexually transmitted disease clinics or using the technique of snowballing, and it is therefore not surprising that the results demonstrated uniformity since prostitutes not contacted by these methods are not represented.

The book is an interesting collection of papers, some anecdotal and others highly scientific, which detail the connection between prostitution and drugs as well as providing some very limited data on HIV before 1989. Although the reports confirm that except for in Africa the majority of HIV in prostitutes is related to injection drug use, it does not answer questions relating to clients, with no new data on whether HIV is being spread by prostitution or whether clients are responding to the risk. I suspect that it will have only a limited audience although it serves the useful purpose of bringing together a diverse collection of reports on prostitution.

R. P. BRETTLE, *Consultant Physician and part-time Senior Lecturer, Infectious Disease Unit and Department of Medicine, City Hospital, Edinburgh*

The Practical Application of Medical and Dental Hypnosis. By MILTON H. ERICKSON, SEYMOUR HERSHMAN and IRVING I. SECTER. New York: Brunner/Mazel. 1989. 480 pp $18.95.

For the serious therapist, fresh accounts of the work of Erickson should always be welcomed. Unfortunately, in this volume Hershman and Secter do him little justice, particularly as a first-named, albeit posthumous author.

The book consists of 11 chapters, with case histories and transcriptions of discussions between therapists and subjects as recorded at various seminars throughout the USA.

In the chapter outlining the history and theory of hypnotism, the former is not entirely accurate and the latter is all too brief. The results of electroencephalographic studies should warrant more than two lines. Thus, the important works of Wyke, Rozhnov and Ulett *et al* are notable for their absence. Current theories of suggestibility and hypnotisability are also omitted, but fascinating interviews by Erickson and others are recorded as a means of illustrating some of the phenomena of hypnosis. The discussion on anaesthesia should have mentioned the use of hypnosis in minor surgery and in plastic surgery, particularly following injury and burns. A brief discussion of the advantages and disadvantages of hypnosis in obstetrics omits useful references to those such as Fuchs *et al* who report extensively on the treatment of hyperemesis.

On page 228, the patient is told that all the information she has ever heard, read or seen "is stored in the subconscious". Is it?

Chapter 7, "Hypnosis in children", is an area in which this therapy can be invaluable. Yet no mention is made of the classical work of the Hilgards on the amelioration of pain, particularly as applied to the treatment of leukaemia.

A hotch-potch of subjects follow. These are discussed under such headings as "Fears", "Pains" and "Therapy of habits", etc., while "Phobias" merit a mere six and a half lines Terminal cancer, upon which so much devoted work has been carried out, would deserve a more sympathetic approach, and the chapter on "Clinical applications of hypnosis in psychiatry" is far from helpful. "Hypnosis in dentistry" is certainly of value but I wonder why the stage hypnotist, the lay use of hypnosis and medicolegal liability are included in this chapter.

Apart from the delightful passages by Erickson, the book has some merits but is far from essential reading.

DAVID WAXMAN, *Associate Specialist in Psychiatry (Retired), Department of Psychological Medicine, Central Middlesex Hospital, London*

Cities Are Good For Us. By HARLEY SHERLOCK. London: Transport 2000. 1990. 99 pp. £10.00.

The reason for reviewing this short book (prelude to an expanded version next year) in a psychiatric journal is that it links the subject clearly with health, and particularly mental health. It contains no specifically medical data, but a great deal of common sense that justifies the title; although it is mostly about Britain, the general principles apply almost everywhere. Sherlock is that rare bird – an architect who is less concerned with the impressive or fashionable appearance of structures than with their suitability for people; he also focusses in a highly practical way on the relationship of buildings to their surroundings and to the transport network. As the former chairman of Transport 2000, an enlightened but largely ignored environmental organisation, he is well able to argue that accessibility, rather than mere mobility, is critical to the life of cities.

Human history shows that people have migrated into cities since these settlements first existed: the flood is greater today than ever before. Yet, as Sherlock says, "cities are now seen, at best, as a great social problem and, at worst, an irrelevance to the twenty-first century". That the urban environment has, on the whole, become worse "is caused largely by unimaginative housing, misguided transport policies and the consequent loss of people and their activities from inner-city areas". The knock-on effect is that surrounding countryside is steadily destroyed by mostly unwilling migrants from the cities, so that both town and country lose out simultaneously.

The usual excuse for not doing what would make people's life better is that there is "no money". Yet the evdence here demonstrates clearly that to organise cities in a more healthy way would actually cost less overall than what we are doing now – even if a minority paid more for their privileges, such as unrestricted motoring. 'Universal' car ownership is largely a myth, yet relatively modest increases in commuting by car have brought city centres almost to a standstill, while at the same time undermining public transport, which is really a city's lifeblood. The same is true in housing: unimaginable sums have been spent on 'comprehensive' redevelopment and the creation of megastructures; these mostly function worse than what they have replaced, and ruin everything that remains around them.

At the end of this impressive tract it is hard to resist the thought that national policies which affect the human habitat are wholly irrational, although short-term economic gains often provide the explanation. Yet only few other countries do better than Britain, as those attending the World Psychiatric Congress in Athens last year will have been well aware. Most ordinary people detest what is happening, but seem powerless to change it.

HUGH FREEMAN, *Editor, The British Journal of Psychiatry*

Examination Notes for the MRCPsych Part I. By BASANT K. PURI and JON SKLAR. London: Butterworths. 1989. 165 pp. £9.95.

No prizes for guessing that the authors have aimed this primarily at the needs of examination candidates. Who else would be so desperate as to choose a text consisting largely of bare headings and lists? I know: psychiatric lecturers and tutors. Wrong: apparently medical students, psychiatric nurses, psychologists and trainee psychotherapists should also find this useful.

For the most part the book works its way conscientiously, chapter by chapter, through the topics specified as the content of the multiple choice questions (MCQ) paper in *General Information and Regulations for the MRCPsych Examinations* (which is essential reading), but in reverse order. The concentration of factual information is impressive, if indigestible, and the sections on neuropharmacokinetics and drug actions and interactions are particularly helpful. The chapter entitled "Methods of clinical assessment in psychiatry" contains (under the subheadings "Qualities of a good history" and "Clinical skills") some golden words of advice. All the greater is then our dismay to find this followed not by a synopsis of the broad topic of explanatory psychopathology but by an exposition of one small part of this – psychoanalytic theory. The unwary reader should be warned that the examiners may not be so blinkered. Valuable space is then squandered on some curiously incongruous dissertations ending with "The sexual life of the patient". The final chapter on classification, also loses its way, distracted by the exotica of psychodynamic nosology.

This book is unlikely to appeal to the collector of MCQ samples – only eight specimens given here, all available elsewhere – but will doubtless find avid readers among those given to the frenzied last minute swot; they will find a compact if not especially well balanced meal here.

DAVID JULIER, *Littlemore Hospital, Oxford*

The Mind: Its Origin, Evolution, Structure and Functioning. An Independent Study of the Basic Mental Processes. By MALCOLM I. HALE. Pittsburgh, USA. Hale van Ruth. 1989. 225 pp. $7.95.

This book is no exception to others on the subject of the mind in that it attempts to grapple with the intricacies and functioning of this most complex human phenomenon. The book embraces four main sections. Part I explores the evolutionary origin of those aspects of the brain functioning which are inextricably bound up with one another to form a functionally single apparatus, the mind. Part II outlines the basic influences determining the evolutionary development of the mind from the approximate level of present-day apes. Part III is concerned with a detailed analysis of the different subfunctions of the mind in terms of the complex relationship between physical structure and psychological function. Part IV examines the basic functioning of the mind with particular reference to the higher processes of language, reasoning, understanding, etc. and the ways in which these processes are generated and stored.

The stance taken by the author is very much a biological one. Hence, he argues: "Such philosophical and psychological ideas of mind suffer from a crucial weakness: they equate the *products* of the apparatus with the apparatus itself. . . . Yet how the brain thinks and how it understands are purely biological processes: the functioning of energy-consuming living tissue" (p. 107). His argument for a biological basis of consciousness makes for quite interesting reading, but as with most books on this challenging subject it raises many more questions than it answers.

TRIAN FUNDUDIS, *Top Grade Clinical Psychologist, Fleming Nuffield Unit, Jesmond, Newcastle upon Tyne*

Management of Normality. By ABRAM DE SWAAN. London: Routledge. 1990. 234 pp. £9.99.

Subtitled *Critical Essays in Mental Health and Welfare*, this book examines how definitions of 'normality', reinforced by reductionist research methodologies, are used to justify 'protoprofessionalisation', a process whereby everyday problems are increasingly seen as the province of specialists. It is argued that society is, by this means, shielded from the suffering, and encouraged to avoid struggling with the political, moral and social issues inherent in the distress of others. De Swaan further dissects the complexities of the relationship between 'helper' and 'help seeker' – how they are interdependent and yet contain inherent conflicts of interest. Processes whereby the various professions stake out and defend their developing territories are examined.

The first section explores these ideas in relation to the medicalisation of extensive areas of life, leading to an increasing preoccupation with physical health and diet rather than, for instance, character formation and social conflict. The existence of a covert agreement between doctor and patient to discuss certain issues and not others is well illustrated in a chapter on "Affect management in a cancer ward". This examines in detail the defensive strategies used to protect both staff and patients from being overwhelmed with anxiety at their shared predicament.

The second section focuses on the complexities of the relationship between psychotherapist and client, and the ground rules and mystique inherent in the process which, it is argued, serve to allocate power and control to the therapist. A final section examines the social contexts of 'agoraphobia', 'jealousy', 'intimacy' and 'the survivor syndrome'.

By exploring 'normality' through the dual perspectives of sociology and psychotherapy, De Swaan has produced a thought-provoking, densely argued and important book which raises issues which deserve to be widely considered and debated, particularly by those in the helping professions. It is well produced and extensively annotated, although a larger typeface would have made it more comfortable to read

MARGARET R. HILTON, *Consultant Clinical Psychologist, Department of Forensic Psychiatry, St George's Hospital, London*

Handbook of Psychooncology: Psychological Care of the Patient with Cancer. Edited by JIMMIE HOLLAND and JULIA ROWLAND. 1989. 785 pp. £85.00.

The development of psychooncology has paralleled the growth of general liaison psychiatry in the United States and considerable clinical and research work has been done in the last 20 years. It is timely and appropriate that a review of this work has now been compiled by psychiatrists and allied mental health professionals at the Memorial Sloan-Kettering Cancer Centre, who are pioneers in this field. Overall, their book is well written and comprehensive. In those areas of interest where there is still a paucity of established data, the authors have provided sensible and practical information based on their clinical experience.

The section on factors which influence psychological adjustment to cancer pays considerable attention to the role of medical variables and rather less to personal ones. This is perhaps surprising when current evidence suggests that patients' psychological response to illness is determined more by their thoughts and beliefs about illness, social support and past history of psychiatric illness than by the symptoms, severity or treatment of the disease itself.

There are useful accounts of the psychiatric disorders associated with cancer and the psychological management of pain, nausea and vomiting, and anorexia. The chapters on therapeutic interventions for psychological morbidity are generally detailed. It is however disappointing that less than half a page is given to consideration of cognitive techniques, when clinical and experimental data increasingly supports their efficacy in the management of mood disturbances in both physically healthy and ill patients.

I found the section on the psychological and ethical dilemmas and stresses faced by different professionals working with cancer patients particularly interesting. These issues are poorly covered in other texts and this account offers valuable insights for those who practise their liaison psychiatry as part of a multidisciplinary team. The interface between psychiatry, law and ethics is given careful consideration in a chapter which focuses on issues such as psychological problems in decision making and the vicissitudes of truth telling.

At the end of the book the role of psychosocial factors in the development and progression of cancer are outlined in a measured and rather terse account. This does insufficient justice to the data in this field and perhaps reflects the fact that the book is written from a single institution. A second edition of the book in the next decade will hopefully reflect the anticipated growth of this area of psychosocial oncology. Current work on the interaction between psychological factors and the central nervous, immune and endocrine systems is particularly exciting. A subsequent edition might also give further consideration to psychological issues related to prevention and early detection of cancer, to the measurement and classification of psychiatric disorder associated with physical illness, and to the outcome of much needed trials of psychological intervention in the management of cancer related psychiatric morbidity.

This book is large and expensive but all those involved in the psychological care of patients with cancer should have access to a copy.

AMANDA J. RAMIREZ, *Lecturer in Psychosocial Oncology, Department of Psychiatry and Imperial Cancer Research Fund Clinical Oncology Unit, Guy's Hospital*

Aggression in Children. Edited by LEA PULKKINEN and J MARTIN RAMIREZ. Seville: Publicaciones de la Universidad de Sevilla. 1989. 155 pp.

There is increasing concern about rates of aggressive behaviour in children and much speculation about its origins. This paperback book contains a collection of eight papers presented at the 1987 Fourth European Conference of the International Society for Research on Aggression.

The first paper is a brief review by Benton of studies of the effects of dietary sugar on children's behaviour. The last paper is a report by the Dutch team of their contribution to the cross-national survey on the effect of viewing television on children's behaviour. This gives a detailed account of the complexities involved in this sort of research, and provides an excellent review and critique of the international study. The other six papers are reports of recently completed or on-going research projects looking at other associations with childrens' aggressive behaviour. For example, two studies consider direct influences such as the modelling of overt violence by family members, and the effect of different forms of parental justification for their own or others' aggressive acts. Other studies from Finland and Holland concentrate on conditions which perpetuate violence, especially peer interactions including bullies' and victims' characteristics and relationships with others. Pulkkinen herself contributes findings from the longitudinal follow-up study of aggressive and anxious children (from age 8 years to 26 years) carried out at the University of Jyvaslyla, Finland.

Those working in academic departments of psychology where there is an interest in aggressive behaviour will already know of the work discussed in this symposium. However, for those who could not go to the meeting and for those clinicians and others who want to hear what is happening on this particular sector of the research front, this small book provides an up-to-date and accessible review.

ROY HOWARTH, *Consultant Psychiatrist, Hospital for Sick Children, London*

Psychiatric Disorders in Children and Adolescents. Edited by BARRY D. GARFINKEL, GABRIELLE A. CARLSON and ELIZABETH B. WELLER. Philadelphia: W. B. Saunders. 1990. 569 pp. £33 50.

This book is described in the foreword as a seminal work, offering a modern approach to the evaluation, diagnosis and treatment of child and adolescent psychiatric disorders It is a North American book, with 30 chapters by different eminent authors, nearly all of whom are psychiatrists. The editors (academic child and adolescent psychiatrists from Minnesota, New York and Columbus, Ohio) have aimed to produce an empirically based clinical textbook, with a laudable ambition to help "child psychiatry to reach the highest standards as a medical speciality". The editors state a further aim to provide a contrast with previous texts which have presented a psychodynamic and behavioural perspective. Moreover, the book is intended to be used as a practical guide, essentially for clinical use rather than for theoretical interest.

Despite the multiple authorship, a consistent style is achieved, each chapter containing mainly useful practical tables and large numbers of case vignettes. The referencing is generally sound and impressively up-to-date, although there is a relative paucity of British references. The layout and presentation are good, making it more readable than many large textbooks.

At times the empirical approach taken seems a narrow one, implying an incompatibility between behaviourism and empiricism and a belief that an empirically sound treatment is a pharmacological one. The style also seems to incorporate an over-confidence in the DSM–III classification system.

The book is arranged in five sections: internalising disorders, disruptive behavioural disorders, developmental disorders, specific clinical issues, and techniques in child and adolescent psychiatry. Although most important areas are covered, and the last section does find some space for individual and family psychotherapies, this arrangement is somewhat idiosyncratic. It is traditional in child and adolescent psychiatry to take a developmental perspective, wherein disturbance is set against the range of normal development. There is no section on this subject, nor is the current understanding of child psychiatric disorder given a historical perspective. Some anomalies result from the organisation of the sections: thus depressive disorder and bipolar disorder are included under internalising disorders, while schizophrenia (given relatively little space) is discussed, in the section on developmental disorders, in a chapter on childhood psychosis. Similarly, while autism and tics are seen as developmental disorders, sleep disorders are contained in the section on specific clinical issues, and attention-deficit disorder in that on disruptive behavioural disorders, where it is afforded two chapters. This compares with one of only 17 pages on conduct disorder (including delinquency).

To conclude, I consider this a worthy, up-to-date, well presented textbook of child psychiatry, although not a fully comprehensive one. It will be of most value to specialist child and adolescent psychiatrists rather than a wider multidisciplinary readership.

SIMON GOWERS, *Senior Lecturer in Child and Adolescent Psychiatry, University of Manchester*

Narcissism: Psychoanalytic Essays. By BELA GRUNBERGER. Madison, Connecticut: International Universities Press. 1990. 311 pp. $19.95.

This is a very specialised area and it behoves writers on such topics to write clearly and to supply a succinct summary of their views, especially where these differ from the orthodox. This paperback was published in America in English in 1990, having been originally published in France and in French in 1971. The foreword was written by a doctor of philosophy whose attachment and profession (?psychologist) are not stated. The book contains essays which were originally published in a French psychoanalytical journal between, if I interpret the author properly, 1956 and 1971.

There are the usual references to the original psychiatric use of the term narcissism as a sexual perversion, the distinction between the psychoanalytical term and auto-eroticism, and Freud's contributions. Grunberger, a Hungarian, tells us that his book is based on prolonged clinical observation. This is not obvious from the text. The latter is highly theoretical and appears to be based more on an analysis of people who never lived outside mythology than on the stuff of the clinical disciplines, real people The problem with narcissism is that it has been seen everywhere (psychosis, neurosis, normals, sleepers, the physically ill) and therefore allows for all sorts of generalisations.

As in another book by this author that I reviewed recently, the style is difficult to read, the text is excessively padded with footnotes and about-turns in mid-sentence, and the reader's patience is is taxed to the extreme. I do not know why this work and the previous one have both hit the English language together in 1990.

BRIAN O'SHEA, *Consultant Psychiatrist, Newcastle Hospital, Greystones, Co. Wicklow, Republic of Ireland*

Psychoanalytic Terms and Concepts. Edited by BURNESS MOORE and BERNARD FINE. Newhaven: American Psychoanalytic Association and Yale University Press. 1990. 210 pp. £25.00.

This mini-encyclopaedia presents both historical and current meanings of the most widely accepted psychoanalytic terms and concepts. It is a revised and expanded version of the earlier glossary of the American Psychoanalytic Association. A substantial editorial board have worked with nearly 200 contributors to produce well informed and precise outlines of various concepts Helpful references are given for further reading.

Looking up several areas of particular interest to me, I found the sections accurate and coherent, avoiding the subtle misunderstandings and distortions that tend to creep in when authors try to write of theoretical areas that are not their own.

There are also sections devoted to particular theorists – e.g. Bion, Klein, Kohut and Winnicott – with outlines of various core concepts. Inevitably the material is somewhat selective; for example, there is no mention anywhere of the important and undoubtedly innovative work of Robert Langs.

Moore provides an introductory essay on the problems of definition in psychoanalysis, discussing, for example, issues of translation, usage in different cultures, and the ambiguity and flexibility of Freud's own writing. Moore argues for the preservation of metaphor in psychoanalytic discourse, while emphasising the need to be aware of distortions inherent in the use of language. He does reveal his American bias for what is new and his dislike of the scholarly tradition in his comments on Laplanche & Pontalis' *Language of Psychoanalysis* (1973). "... invaluable to scholars in that they precisely chart the psychoanalytic port from which various international movements have embarked; unfortunately some of the conceptual vessels are of World War 1 vintage and may sink under scrutiny". This new book successfully maps the contemporary field and is a very valuable reference text, but in my view Laplanche & Pontalis' earlier work is the more essential.

PHIL MOLLON, *District Psychologist and Adult Psychotherapist, Lister Hospital, Stevenage*

Males with Eating Disorders (Eating Disorders Monograph Series, No. 4). Edited by ARNOLD E. ANDERSON. New York: Brunner/Mazel. 1990. 288 pp. $29.95.

This volume, the latest edition to the Eating Disorders Monograph Series, lives up to the high standard set by the preceding volumes and presents up-to-date, authoritative contributions from well known researchers in the field. In its twelve chapters it comprehensively covers eating disorders in males. These conditions have long since been overshadowed by their female counterparts and at times have been ignored or denied, in spite of the earliest descriptions being of anorexia nervosa in both men and women. In reviewing the history of anorexia nervosa in the male, Chapter 1 reminds us of these earliest case reports from Richard Morton in 1689, Robert Whytt in 1764 and Robert Willan in 1790.

It might be argued that there is no valid reason for devoting a monograph to eating disorders in males in view of its accepted similarity to that in females. However, this book more than justifies its existence. By solely focusing on males, an opportunity is created to look at the specific factors for males in terms of sociocultural pressures, self-image problems and sexuality. Chapter 2 breaks new ground by presenting an autobiographical account by Ralph Wilps of his case history of bulimia nervosa. This sets a clear contrast between the almost commonplace accounts by females suffering from eating disorders. Chapters 3, 4 and 5 deal with sociocultural factors, sexuality and body image, contrasting the difficulties of those with eating disorders with the normal male population.

The volume is well structured, being divided into four sections. The first section I have described above while the second section looks at the clinical psychometric studies of male eating disorders. The third section covers treatment and outcome. The final section consists of a chapter by Anderson, the editor, and attempts to integrate the evidence presented in the rest of the book. I can commend this chapter for its well argued discussion of a proposed mechanism underlying eating disorders. Using the philosophical methods of both Kuhn and Popper, Anderson argues that anorexia nervosa and bulimia nervosa are examples of behavioural disorders of motivated behaviour, which have an underlying mechanism of operant behavioural conditioning. He suggests stages of development for both anorexia nervosa and bulimia nervosa. I found his arguments to be clear and compelling.

Overall, the book is well written by its 20 authors and holds one's concentration without effort throughout its 288 pages. It is comprehensively referenced, up to date and must be strongly recommended to those clinically interested in the field of eating disorders.

NEIL L. HOLDEN, *Consultant Psychiatrist and Senior Lecturer, Mapperley Hospital, Nottingham*

The Bad Object: Handling the Negative Therapeutic Reaction in Psychotherapy. By JEFFREY SEINFELD. 1990. 336 pp. $40.00.

The author of this densely written book starts off with the confident assertion that it "shows how to help the patient overcome what has been described by Freud as the most serious obstacle to psychotherapy: the negative therapeutic reaction". He deals predominantly with borderline patients and extensively reviews the literature on negative therapeutic reactions from Freud to Kohut and Kernberg, although he frequently takes issue with the latter two, and draws his ideas most strongly from Searles, Fairbairn and Mahler. In particular he uses Searles' concept of the out-of-contact phase of therapy where the patient and the therapist are isolated in their own psychic territories until the patient gradually permits the therapist entry into his psychic space. Gradually the patient reaches the phase of ambivalent symbiosis where he or she has enough of a positive self and object representation unit to project a hoped for good object into the transference.

In general, the theoretical ideas are clearly presented, although at times there is the problem of overlap of ideas with different terminology. However, it is in the personal case histories that the author really comes into his own, following them through over a number of years, although at times I did not feel the case example was necessarily an example of the theoretical point being considered. This did not seem to matter too much, although purists might dispute this. In cases which the author had supervised however there was a problem with sounding too dogmatic at times, but perhaps it is easier to be modest about one's own cases than second-hand ones. Towards the end the book rather ran out of steam; most of the interesting ideas had been expanded upon in the first two-thirds and the book could have been shorter without losing its essence. One bone of contention: the author frequently turned projective identification into a verb, for example, "she imagined herself to be rejected by projectively identifying the rejecting internal object into him" (p. 81). Whether or not this is an example of projective identification, which I would not consider it to be, it is clearly grammatically incorrect and occurs with irritating frequency. These quibbles apart, this book is worth a read for the case descriptions alone.

TERESA BLACK, *Specialist Senior Registrar in Psychotherapy, Department of Psychotherapy, Manchester Royal Infirmary*

Sigmund Freud and Art: His Personal Collection of Antiquities. Introduction by PETER GAY. London: Thames and Hudson. 1989. 192 pp. £18.95.

That a doctor who started his career almost penniless and raised a large family should also have accumulated some 2000 objects of antiquity (not to mention a library of 2500 volumes) needs some explaining. A few of the sculptures turned out to be fakes and some were presents, particularly in the later years, but the overwhelming majority of this antique hoard were bought by Freud out of his earnings, which were never enormous. How did he do it, and why?

In practical terms, the prices of ancient objects were then a fraction of today's; scientific archaeology was still in its infancy and the international market had scarcely been fuelled by American museums or Japanese banks. Freud began his collection quite early, was a shrewd buyer, and never stopped until his late seventies. But one of its oddest aspects was that the whole was concentrated in his two personal rooms, where every surface was crammed; not a single piece was kept in the family's accommodation. This narcissism – like Freud's travelling first class, while the family went third – has often been described, but rarely interpreted

Colour illustrations in this volume of 87 of the finest pieces convey both the artistic quality and historical importance of the collection, which is now housed at the Freud Museum in London, after a hair's-breadth escape (like its owner) from the Nazis. There are accompanying essays on the analogy of archaeology and psychoanalysis, the legacies of antiquity for psychoanalysis, science and art in Freud's life and work, and the origins of the collection. The Introduction, by Peter Gay, states that collecting preserves early erotic pleasures and is a way of controlling and commanding the world, but that we have not yet penetrated the full meaning for Freud of his antiquities. No doubt compulsive collectors will continue to ponder on these statements. Meanwhile, this is a worthy record of a second legacy left us by Freud – not as great as a new view of the human mind, but still significant.

HUGH FREEMAN, *Editor, British Journal of Psychiatry*

University of London
British Postgraduate Medical Federation
Institute of Psychiatry

Drugs, Alcohol and Tobacco: Making the Science and Policy Connections

A meeting organised by Action on Addiction at The Institute of Psychiatry, London, 16–19 July, 1991

This highly important international meeting will be of interest to research workers, policy makers, clinicians and educationalists. It will mark the inauguration of the National Addiction Centre and the formal opening of its Addiction Sciences Building.

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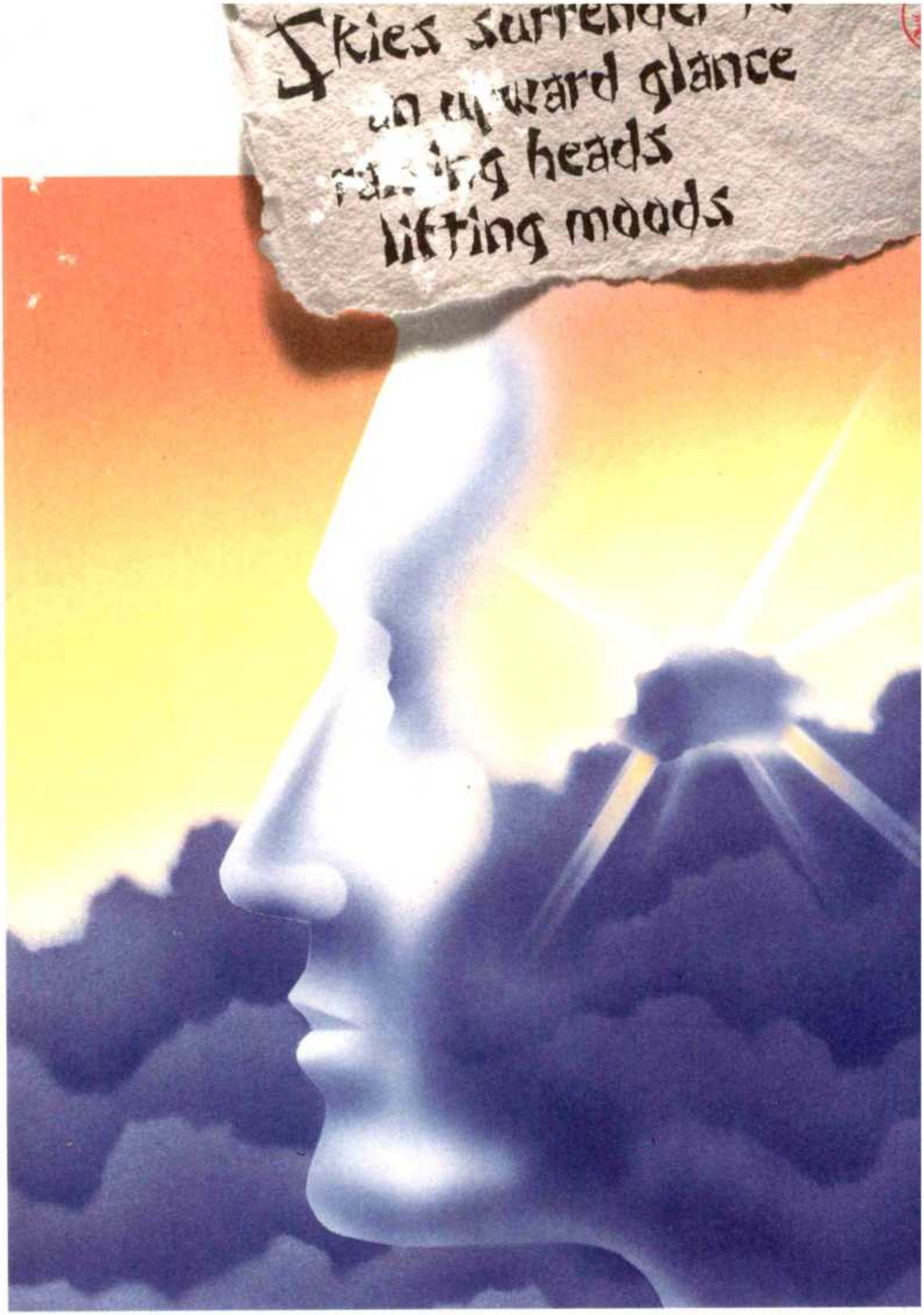

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British Journal of Psychiatry (1991), **158**, 155–157

Monthly and Seasonal Variation in Parasuicide
A Sex Difference

GEORGE MASTERTON

In a total of 22 169 parasuicides over 19 years, there was evidence for a seasonal cycle in parasuicide admissions among women, with an increase during the summer and a fall over the winter, including a pronounced December nadir. Further analysis of the winter trough suggests a sociological explanation, the focal point being Christmas There was no evidence for monthly or seasonal variation among men.

Suicide has long been recognised to have seasonal variation, peaking in the spring and early summer and troughing over the winter (Durkheim, 1897; Swinscow, 1951). This pattern has been confirmed in suicides involving poisons but not in accidental or undetermined poisoning deaths (Barraclough & White, 1978*a*). Sex differences have been reported in the seasonal or monthly distribution of suicide (Meares *et al*, 1981; Nayha, 1982; Parker & Walter, 1982), although not in all studies (Lester & Frank, 1988).

Monthly and seasonal variation in parasuicide (non-fatal deliberate self-harm), analysed by sex, is the subject of this investigation.

Method

Edinburgh has an established database on parasuicides in and around the city, focusing upon a single admission unit, the Regional Poisoning Treatment Centre at the Royal Infirmary (Kreitman, 1977).

For the purposes of this investigation, all parasuicide admissions between 1 January 1969 and 31 December 1987 involving people residing in Edinburgh who were 16 years of age or older were included. These 22 169 events constituted 71% of the total admissions, but this provided a more reliable sample as there have been fluctuations in referrals of adolescents and people living outside Edinburgh.

The data were divided by sex and aggregated for each month. Seasons were defined as three-monthly periods,

Table 1
Aggregate of parasuicide admissions by month and season for years 1969–87 by gender

	Male admissions			Female admissions		
	Expected	Observed	% difference	Expected	Observed	% difference
Month						
January	728	696	−4%	1145	1099	−4%
February	663	673	+2%	1043	1090	+5%
March	730	753	+3%	1147	1124	−2%
April	706	697	−1%	1111	1088	−2%
May	731	718	−2%	1149	1182	+3%
June	708	668	−6%	1113	1182	+6%
July	732	751	+3%	1151	1199	+4%
August	733	770	+5%	1151	1217	+6%
September	710	698	−2%	1115	1159	+4%
October	734	694	−5%	1154	1138	−1%
November	711	751	+6%	1117	1135	+2%
December	736	754	+2%	1155	933	−19%
Season						
Spring	2167	2168	0	3407	3394	0
Summer	2173	2189	+1%	3415	3598	+5%
Autumn	2155	2143	−1%	3386	3432	+1%
Winter	2127	2123	0	3343	3122	−7%
Goodness of fit						
Monthly	$\chi^2=12.4$, d.f. 10, NS			$\chi^2=60.7$, d.f. 10, $P<0.001$		
Seasonally	$\chi^2=0.19$, d.f. 3, NS			$\chi^2=25.1$, d.f. 3, $P<0.001$		

Table 2

Aggregate of parasuicide admissions by week for years 1969–87 in women (November–February) and men (December–January)

	Week													
	Nov 6–12	Nov 13–19	Nov 20–26	Nov 27 -Dec 3	Dec 4–10	Dec 11–17	Dec 18–24	Dec 25–31	Jan 1–7	Jan 8–14	Jan 15–21	Jan 22–28	Jan 29 -Feb 4	Feb 5–11
Women														
No. of admissions	262	281	280	229	230	201	193	213	289	217	227	256	273	276
% difference from expected	+0.5	+7.8	+7.4	−12.2*	−11.8*	−22.9***	−26.0***	−18.3***	+11.8	−16.1**	−12.2*	−1.4	+5.6	+6.7
Men														
No. of admissions	–	–	–	–	184	168	170	158	170	167	151	–	–	–
% difference from expected	–	–	–	–	+10.7	−4.9	+2.3	−4.9	+3.4	+1.6	−8.2	–	–	–

$*P<0.05$, $**P<0.01$, $***P<0.001$.

spring covering March to May and so on. Long-term trends were taken into account by least-squares regression of monthly totals by each sex, with expected values for individual months being calculated from the equation for the best-fitting line.

Observed totals were tested for goodness of fit against expected by χ^2 test. The main monthly finding was further analysed through calculation of the percentage difference between observed totals for weekly intervals and the mean expected value for a week in that month, and this was evaluated statistically by construction of the individual 95% confidence intervals for the difference.

Results

The expected and observed numbers of parasuicide admissions for each month and season are presented in Table 1. There was no pattern of admissions nor significant differences from expected values among men. However, there was evidence for a seasonal cycle among women with above expected numbers from May to September and a fall over winter including a distinct drop in December (−19.2%, 95% confidence interval −25% to −14%).

When the December drop in female parasuicide admissions was examined further the trough was found to occupy eight weeks from the beginning of December with the nadir occurring in the week immediately before Christmas (Table 2). Only the first week of the New Year broke the sequence, the excess almost reaching statistical significance (+11.8%, 95% confidence interval −1% to +25%). Indeed, the three busiest consecutive days throughout these 14 weeks were 2–4 January, with 145 admissions.

The Christmas period was also examined in men, in case the unremarkable monthly results concealed interesting findings. However, Table 2 confirms this was not so. The three busiest consecutive days over these seven weeks were 5–7 January, with 85 admissions.

Discussion

The main findings are evidence for a seasonal variation in female parasuicide admissions, with a sharp winter fall extending for eight weeks around Christmas, contrasting with no monthly or seasonal variation in males.

The obvious explanation for the December nadir is sociological, reflecting the greater impact that Christmas exerts upon women, who traditionally bear the brunt of the responsibility for planning and organising what remains the main family occasion in the year. This may produce an enhanced sense of belonging which could protect some women who are at risk of parasuicide. That this protective effect persists for several weeks after Christmas is in keeping with the findings for parasuicides following World Cup football finals (Masterton & Strachan, 1987) and general elections (Masterton & Platt, 1989), and indicates a deeper mechanism than simply a knee-jerk reaction to an important national event.

The immediate effects of national holidays on suicides have been investigated in the United States, where a reduction in the suicide rate is apparent in the few days before as well as during all holidays (Phillips & Wills, 1987). However, a division emerges during the days immediately after the holiday, with Christmas belonging to the group with a continuing lowered suicide rate while New Year's Day is included in a second group which demonstrates a spike in suicides. Interestingly the Christmas–New Year findings among parasuicides follow the same pattern, especially in women.

Another similarity between the female parasuicide findings and published suicide data is the evident seasonal variation with a winter trough, although the configuration and precise timing of the cycle does differ among these reports (Durkheim, 1897; Swinscow, 1951; Barraclough & White, 1978*b*; Meares *et al*, 1981; Nayha, 1982). Further, when suicides due only to poisoning are considered, the greatest monthly deviation from that expected is also the December reduction (−14.7%) rather than the peak in April/May (Barraclough & White, 1978*a*).

A major difference between parasuicide and suicide is of course the absence of recognisable biological mental disorders in most parasuicide patients, so the demonstration of seasonal variation among parasuicide women that resembles the annual cycle in the suicide rate supports the sociological or Durkheimian explanation against a primary biological determination. The negative findings in male parasuicides suggests that there may be important differences between the sexes in the impact upon people's lives that is made by social or national factors which follow an annual course.

Acknowledgement

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British Journal of Psychiatry (1991), **158**, 158–164

A Magnetic Resonance Imaging Study of Schizophrenia: Brain Structure and Clinical Symptoms

A. H. YOUNG, D. H. R. BLACKWOOD, H. ROXBOROUGH,
J. K. McQUEEN, M. J. MARTIN and D. KEAN

Thirty-one patients with schizophrenia and 33 normal control subjects underwent MRI. The BPRS was used to rate clinical symptoms and the NART to estimate pre-morbid IQ. All were right handed. The temporal lobe was significantly smaller on the left than the right in both the control and schizophrenic groups. The amygdala was smaller on the left than the right in controls but not in schizophrenics. The parahippocampal gyrus was smaller on the left side in the schizophrenic group but not in controls. In the schizophrenic group, ventricular enlargement and cerebral atrophy were significantly related to severity of symptoms. Patients with marked negative symptoms had a bilateral reduction in the size of the head of caudate and the two measures were significantly correlated. Patients with marked positive symptoms had larger VBRs and again the clinical and morphometric changes were significantly correlated. There were no morphometric differences between patients with short duration (two years or less) and chronic symptoms.

Structural changes in the brains of schizophrenic patients have been sought ever since the clinical syndrome was first described. Alzheimer (1897), Kraepelin (1919), Bleuler (1930) and Freud (1964) all thought that brain disease caused the clinical symptoms. Early work suggested ventricular dilatation and cortical atrophy in the brains of chronic schizophrenics from both post-mortem and pneumoencephalographic evidence (Southard, 1915; Haug, 1962). The development of the computerised tomography (CT) permitted relatively non-invasive investigation of brain structure. Following the first study (Johnstone *et al*, 1976) a large number of studies using CT have reported quantitative differences between schizophrenics and controls. Dilatation of the lateral and third ventricles and cortical atrophy have been the commonest findings (Revely, 1985; Weinberger *et al*, 1983). Negative symptoms and cognitive impairment have been associated with ventricular enlargement (Johnstone *et al*, 1976; Andreasen *et al*, 1982).

Magnetic resonance imaging (MRI) offers certain advantages over CT, including superior resolution between grey and white matter and the capacity to image in the coronal plane. Using MRI, Besson *et al* (1987) found that the area of the lateral ventricles was enlarged in the coronal plane, whereas Smith *et al* (1987) found no difference in the area of the third or lateral ventricles in schizophrenics when compared with controls.

In the present study we used MRI to compare brain structure in schizophrenics with normal controls, and examined the relationship between clinical symptoms and brain structures.

Method

Thirty-one patients consented to take part in the study. All satisfied Research Diagnostic Criteria (RDC; Spitzer *et al*, 1978) for schizophrenia and this was based on individual interview, review of the case notes, and discussion with the clinician in charge of the patient. All patients were receiving therapeutic doses of antipsychotic medication. None of the participants had a family history of schizophrenia, or a history of head injury, substance dependence, or a known neurological condition, and all were unequivocally right handed as measured by the Handedness Scale (Annett, 1970).

All patients were rated for clinical symptoms at the time of scanning using the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962) and the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1967). The withdrawal retardation subscores of the BPRS (emotional withdrawal, motor retardation and blunted affect) were taken to indicate negative symptoms, and the thinking disturbance subscores on the BPRS (conceptual disorganisation, hallucinatory behaviour, unusual thought content) were used to rate positive symptoms. Pre-morbid intelligence was estimated using the National Adult Reading Test (NART). This is a reliable measure of pre-morbid intelligence and educational level (Nelson, 1982). The score on this test was converted to an equivalent IQ in the range 87–128.

A control group of 33 healthy volunteers who were matched for age and sex with the patient group was recruited from hospital staff, students, and members of the local community. All controls were unequivocally right handed and none had a personal or family history of psychiatric illness, or a history of head injury, substance dependence or a known neurological condition.

The MRI scans were performed using the same procedure on all subjects. An M&D Technology Limited 0.08 T resistive MRI system was used and two series of scans were performed. In a preliminary study a pulse sequence was chosen that produced the clearest images of medial temporal lobe structures. For this high-resolution scan, a saturation–recovery pulse sequence with a repetition time (TR) of 800 ms and a reduced flip angle (60°) was used. Matrix size was 256^2, slice thickness was 8 mm, and six averages were performed. In the same preliminary study the settings which gave the clearest images of the ventricular system were identified. The low-resolution scan used a saturation–recovery pulse sequence with a TR of 500 ms and a flip angle of 90°. Matrix size was 128^2, slice thickness was 12 mm, and one average was done. Ten serial coronal sections were performed first at the low-resolution settings then the high-resolution settings, with the subjects' head held still in a light headrest. The coronal sections began at the anterior of the temporal lobes and extended posteriorly.

All measurements were done without knowledge of subject identity or of group membership. The MRI images were fed to a Quantimet 800 Image Analysis System (Cambridge Instruments Ltd) via a Plumbicon scanner, from films placed on a transilluminating macroviewer. The analogue image acquired by the Quantimet was converted to a digital matrix. Areas for measurement were outlined on the monitor screen using a light pen and the data on them extracted from the digital matrix. To ensure consistency, five repeated measurements of each area were made and the mean taken.

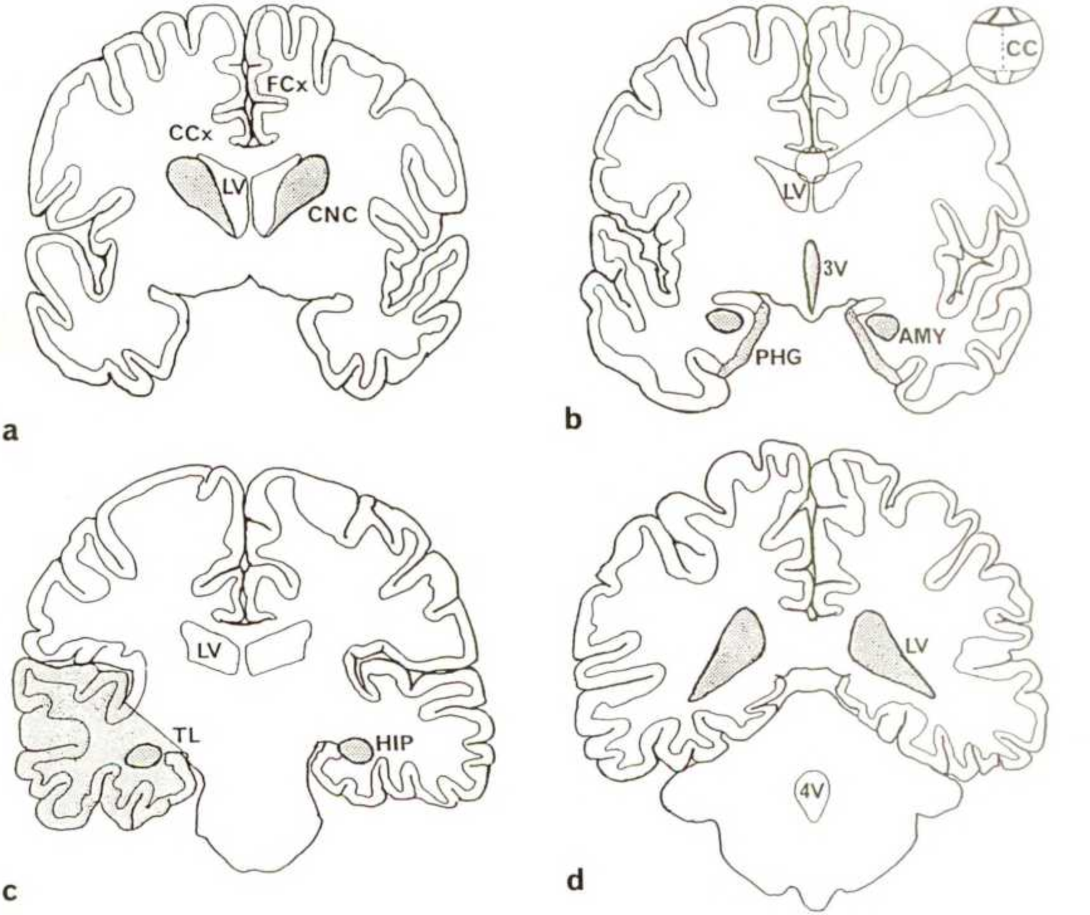

Fig. 1 Illustrations adapted from Nienwerhuys *et al* (1981) showing coronal sections through: (a) rostral to anterior commissure and optic chiasm, (b) intraventricular foramen, infundibulum and amygdaloid body, (c) thalamus, cerebral peduncle and pons, (d) splenium of corpus callosum and fourth ventricle. The inset to (b) shows where the thickness of corpus callosum was measured. Areas measured in this study were: AMY, amygdala; CC, corpus callosum; CCx, cingulate cortex; CNC, head of caudate nucleus; FCx, frontal cortex; HIP, hippocampus; LV, lateral ventricle; PHG, parahippocampal gyrus; TL, temporal lobe; 3V, third ventricle; 4V, fourth ventricle.

The reliability of the measurements made was checked in the following manner: 13 films were selected randomly from the total of 64. Both low-resolution and high-resolution scans were examined, and selected areas were measured five times. Coefficients of variation were calculated for these areas. On the low-resolution scan the coefficient of variation for the measurement of the lateral ventricles was 6.9%. The coefficient of variation for measurements on the high-resolution scans were less than 8%, except for the right amygdala which was 10.8% and the left amygdala which was 10.2%. Repeated measurements over the course of the study indicated no significant operator difference in measurement. The measurements were given in mm and mm^2 of the actual photographic image.

The brain areas studied are shown on Fig. 1. The areas were selected for measurement using an anatomical atlas (Nienwerhuys *et al*, 1981) and the images selected which corresponded most closely to Fig. 1. Sample images are shown in Fig. 2. Measurements were made on both the low-resolution scan and the high-resolution scan. The total intracranial area (ICA) was taken as the area delineated by the inner table of the skull. A straight line was extrapolated across the lower margin, occupied by the brainstem, to complete the delineation. The cerebral size (CS) was taken as the area delineated by the surface of the brain at the level of the amygdala, measured from the medial aspect of the temporal cortex around the surface of the brain to the opposite medial aspect of the temporal cortex. A straight line was extrapolated to connect the two opposing

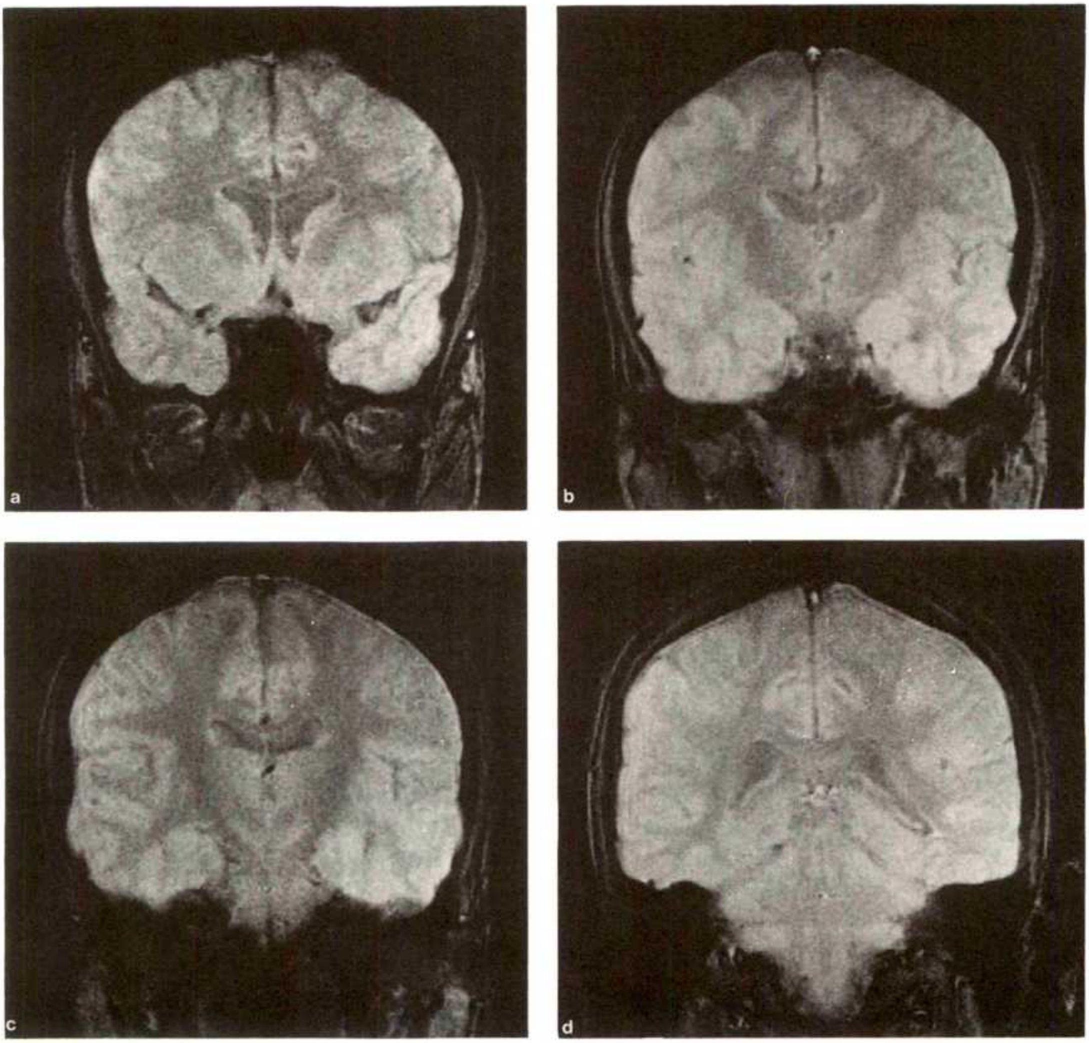

Fig. 2 High-resolution MRI scans corresponding to sections outlined in Fig. 1.

Table 1
Mean (s.d.) brain areas (expressed as a ratio of intracranial area, × 100)

Area	Controls (no. of scans[1])	Schizophrenics (no. of scans[1])	Mean difference (95% confidence interval)
Third ventricle	0.36 (0.14) (33)	0.40 (0.12) (30)	0.04 (0.035, 0.43)
Ventricle : brain ratio	291 (170) (33)	315 (141) (31)	24 (−27.4, 74.2)
Amygdala			
right	0.40 (0.14) (22)	0.39 (0.15) (22)	0.01 (0.002, 0.014)
left	0.38 (0.13) (20)	0.40 (0.15) (22)	0.02 (0.011, 0.023)
Parahippocampal gyrus			
right	0.30 (0.09) (23)	0.30 (0.10) (24)	0.00
left	0.30 (0.07) (23)	0.29 (0.10) (25)	0.01 (−0.08, 0.05)
Hippocampas			
right	0.56 (0.17) (31)	0.62 (0.18) (31)	0.06 (0, 0.12)
left	0.54 (0.17) (32)	0.59 (0.16) (31)	0.05 (−0.01, 0.11)
Caudate nucleus			
right	1.08 (0.22) (33)	1.15 (0.29) (31)	0.07 (−0.03, 0.17)
left	1.06 (0.23) (33)	1.11 (0.28) (31)	0.05 (−0.05, 0.15)
Temporal lobe			
right	11.50 (1.60) (30)	10.90 (1.30) (31)	0.6 (0.1, 1.1)
left	10.70 (1.40) (30)	10.30 (1.40) (31)	0.4 (−0.1, 0.9)
Cingulate area			
right	1.02 (0.24) (25)	0.95 (0.21) (26)	0.07 (−0.03, 0.17)
left	1.06 (0.27) (25)	0.96 (0.20) (26)	0.1 (0, 0.2)
Frontal area			
right	0.75 (0.18) (25)	0.78 (0.19) (26)	0.03 (−0.05, 0.11)
left	0.74 (0.19) (25)	0.82 (0.21) (24)	0.08 (0, 0.16)
Thickness of the corpus callosum : mm	14.8 (2.0) (33)	14.4 (3.0) (31)	0.4 (−0.24, 1.0)
Atrophy . mm[2]	123 (11) (33)	124 (8) (31)	1 (−0.3, 2.3)

1. Some areas were not possible to determine on all scans.
2. Calculated as the difference between the ICA and CS measured on the same slice.

medial temporal cortices. The ICA and CS for each subject were measured on the low-resolution scan at the level of Fig. 1(c).

Cerebral atrophy was calculated for each subject from the difference between CS and ICA. All other measurements were converted into a ratio relative to ICA for that subject. The ventricle : brain ratio (VBR) was taken from the section of the low-resolution scan in which the lateral ventricles had the greatest area (Fig. 1(d)); the right and left sides were added together to give a total area which was then expressed as a ratio of the ICA to give the VBR. All other measurements were carried out on the high-resolution scan as shown on Fig. 1.

Results

The patient group comprised 24 men and seven women (mean age 29.3 years, range 20–50), with a mean duration of illness of 70 months (range 1–390) and a mean IQ of 110 (range 91–125). The 25 male and eight female controls (mean age 28.3 years, range 21–52) had a mean IQ of 121 (range 110–127). The ratios of brain areas to ICA are given in Table 1. In both the schizophrenic and control groups, and in all areas, these ratios conformed to a normal distribution (verified by the Kolmogorov–Smirnov test).

Between-group comparisons of all brain areas using Student's two-tailed t-test revealed no significant differences between controls and schizophrenics. However, the NART score was significantly greater in controls compared with schizophrenics ($t = 5$, d.f. $= 62$, $P < 0.001$). Because of a possible effect of IQ, analysis of covariance with age and NART as covariates was carried out. However, there were no significant correlations (at the 0.05 level of significance using Pearson's correlation) between brain regions and NART score or age in schizophrenics or controls.

Paired t-tests were used to compare the right and left measurements of the amygdala, hippocampus, parahippocampal gyrus, caudate, cingulate, the temporal lobe, and the lateral ventricles. In both the control and schizophrenic groups the temporal lobe was significantly smaller on the left compared with the right (paired t-tests: for schizophrenic group $t = 3.2$, d.f. $= 30$, $P < 0.005$; for control group $t = 5.7$, d.f. 29, $P < 0.001$). The size of the right–left difference in the temporal lobes did not differ between the groups, indicating that both the schizophrenics and control group had a similar right–left asymmetry of the temporal lobe measurement. The left amygdala was smaller than the right in controls ($t = 3.0$, d.f. 19, $P < 0.01$) but not in schizophrenics when compared with paired t-test. The absolute right–left difference between controls and schizophrenics for the amygdala was significant ($t = 2.03$, d.f. 41, $P < 0.05$). In the schizophrenic group, but not in controls, the parahippocampal gyrus was smaller on the left than on the right ($t=2.3$, d.f. 23, $P<0.05$). However, the absolute right–left difference between the two groups was not significant.

The Pearson's correlations of clinical variables with selected MRI measurements were examined. The relationship between positive symptoms (BPRS thinking disturbance subscore), negative symptoms (BPRS withdrawal retardation subscore) and the total symptoms (total BPRS) was examined. There was a significant correlation between the score for positive symptoms and VBR ($r=0.49$, $P<0.005$). No other significant correlations were found with positive symptoms. A significant inverse correlation was found between the score for negative symptoms and the size of the caudate nucleus on both the left side ($r=-0.45$, <0.01) and the right side ($r=-0.4$, $P<0.03$).

For further analysis the schizophrenic group was subdivided into those with negative symptoms (a score greater than 2 on the BPRS withdrawal retardation subscore) (group 1, 16 patients) and those having no negative symptoms (a score of 2 or less on the BPRS withdrawal retardation subscore) (group 2, 15 patients). In group 1 the caudate nucleus was smaller than in group 2 on the right ($t=3.4$, d.f. $=29$, $P<0.002$) and the left side ($t=3.8$, d.f. 29, $P<0.001$). No other significant differences were found.

Positive symptoms were examined in the same manner. The schizophrenic group was divided on the basis of scores on the thinking disturbance subscore of the BPRS: group 1 ($n=12$) had scores greater than 2 and group 2 ($n=19$) had scores equal to or less than 2. The right caudate nucleus was significantly smaller in group 1 than in group 2 ($t=2.6$, d.f. $=29$, $P<0.02$). The VBR was significantly greater in group 1 than in group 2 ($t=2.45$, d.f. $=29$, $P<0.02$). No other significant differences were found.

A total symptom score for the schizophrenic group was taken from the total BPRS rating. In group 1 ($n=15$) the total BPRS score was equal to or greater than 5, in group 2 ($n=16$) it was less than 5. The hippocampus on the right was smaller in group 1 than in group 2 ($t=2.61$, d.f. $=20$, $P<0.02$). The caudate nucleus was smaller in group 1 when compared with group 2 on both the right ($t=3.20$, d.f. $=21$, $P<0.004$) and the left side ($t=3.67$, d.f. $=24$, $P<0.001$).

The schizophrenic group was similarly divided on the basis of duration of illness into two groups. Group 1 ($n=15$) included patients whose illnesses started less than two years previously, group 2 ($n=16$) included patients with a duration and illness greater than two years. No difference was found in any measure between these groups, including NART scores.

Discussion

In this study the temporal lobe area on coronal section was significantly smaller on the left than the right in both schizophrenics and controls. Similarly, in controls the amygdala was smaller on the left than the right, whereas in the schizophrenic group this asymmetry was not present. The parahippocampal gyrus was smaller on the left side in the schizophrenic group only. Greater negative symptoms and a greater overall symptom score were both associated with smaller caudate nuclei, and greater positive symptoms were associated with an increase in VBR. No association was found between age, NART score or duration of illness and any measures of brain area.

Multiple comparisons were made in this study and this raises the possibility of significant results arising due to chance. No significant differences were found when comparing brain areas between schizophrenics and controls, despite the large number of comparisons. The right–left differences were examined in 14 comparisons, raising the possibility of a significant result due to chance at the 0.05 level. Four significant findings resulted from these comparisons (temporal lobe smaller on the left than right in controls and schizophrenics, the amygdala smaller on the left in controls and the parahippocampal gyrus smaller on the left in the schizophrenic group). Similarly, because of the multiple comparisons of clinical variables with MRI measurements (36 comparisons in total), two significant results would be expected to arise by chance at the 0.05 level. Seven significant correlations were found.

Asymmetry of the planum temporale and Sylvian fissure, two temporal lobe structures, has previously been reported in normal subjects (Geschwind & Galaburda, 1985). The planum temporale is usually larger on the left side whereas the Sylvian fissure rises more steeply and extends further back on the left. The temporal lobe has previously been reported to be larger on the right than the left (LeMay & Kido, 1978; Jack *et al*, 1988) in normal subjects. DeLisi *et al* (1988) investigated brain limbic structures using MRI in schizophrenics who had a family history of schizophrenia and found evidence of significant reduction in the size of medial temporal lobe structures in schizophrenics compared with controls. Kelsoe *et al* (1988) estimated the volume of various brain structures using sequential coronal MRI scans. They found significant increases in the volume of the lateral ventricles and the area of the third ventricle in the schizophrenic group but no difference in other brain structures. Suddath *et al* (1989) reported the left temporal lobe to be smaller than the right in schizophrenia. Suddath *et al* specifically examined the volume of grey matter in schizophrenics and controls and reported significantly smaller volumes bilaterally in the temporal lobes of schizophrenics corresponding to the location of the amygdalae and anterior hippocampi. Johnstone *et al* (1989) measured temporal lobe structure using MRI in schizophrenia and bipolar affective disorder. They reported the temporal lobe area being less on the left when compared with the right in schizophrenia but not in bipolar affective disorder or controls. It must be emphasised that although these studies have found statistically significant differences this does not necessarily mean that these differences are anatomically significant.

Our findings are also in keeping with recent neuropathological research (Roberts & Crow, 1987). Reynolds (1983) found a specific increase of dopamine in the left amygdalae of post-mortem schizophrenic brains. Bogerts *et al* (1985), Brown *et al* (1986) and Falkai *et al* (1988) report abnormal morphology and shrinkage of temporal lobe structures including the amygdala and parahippocampal gyrus in post-mortem schizophrenic brains. Reynolds (1989) has recently suggested that a deficit and/or disturbance of neurones in temporal limbic regions could lead to a disinhibition of limbic dopamine neurones, providing the means whereby neuroleptic drug treatment might ameliorate the effects of a neuronal deficit in schizophrenia.

Since the first of the modern brain imaging studies by Johnstone *et al* (1976) there has been speculation about the relationship between clinical symptoms and lateral ventricular enlargement. Early studies suggested an association between lateral ventricular enlargement and negative symptoms (Johnstone *et al*, 1976; Andreasen *et al*, 1982). However, Farmer *et al* (1987) found that patients with positive symptoms had larger ventricles than those with mixed positive and negative symptoms. Our findings are in keeping with this, as we found a significant positive correlation in the schizophrenic group between enlarged lateral ventricles and greater positive symptoms. The failure to find a correlation between the duration of illness and VBR is in keeping with previous reports (Weinberger *et al*, 1983) and suggests that brain changes may precede the onset of clinical symptoms. Our finding of smaller caudate nuclei associated with a greater degree of negative symptoms could be an effect of medication. However, abnormalities in the globus pallidus, which is functionally related to the caudate nuclei, have been reported in never-medicated schizophrenics using positron emission tomography (Early *et al*, 1987).

It is now accepted that the brain is abnormal in schizophrenia. We have demonstrated a distinct pattern of quantitative structural abnormalities in the limbic system of living schizophrenic patients using MRI. Furthermore we have shown that quantitative changes in brain structure are associated with more severe clinical symptoms.

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Morbidity Risks of Schizophrenia and Affective Disorders among First-Degree Relatives of Patients with Schizoaffective Disorders

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Of 510 patients consecutively admitted and diagnosed as schizophrenic, 310 who failed to meet research criteria for schizophrenia were labelled as having 'atypical psychosis'. This heterogeneous group of patients was then subtyped into more homogeneous subgroupings according to their clinical characteristics, independent of their family data. One subgroup resembled schizophrenia, one resembled affective disorders and a third ($n = 57$), which did not resemble either schizophrenia or affective disorder, was defined as 'schizoaffective'. Comparing the morbidity risks for schizophrenic and affective disorders in the relatives of this schizoaffective group with those of the relatives of 'typical' groups of schizophrenia and affective disorder, showed that this group was different from those with schizophrenic and affective disorders.

This paper investigates the morbidity risks for schizophrenia and affective disorder among the relatives of 'schizoaffective' patients. It was hoped that such a study would tell us more about where schizoaffective disorder stands in relation to the major psychoses, since there is no consensus concerning the nosological status of schizoaffective disorder. Kasanin, by introducing the term 'schizoaffective psychosis' in 1933 to describe a psychotic condition characterised by sudden, acute onset, good pre-morbid functioning, both affective and schizophrenic symptoms, and usually complete recovery, spurred new interest in delineating between the major psychoses. However, the nature of schizoaffective disorder still remains elusive and controversial.

Following Bleuler's example, atypical cases of schizophrenia, with features of manic–depressive psychosis, have sometimes been grouped together and considered a subtype of schizophrenia (Coble, 1953; Croughan *et al*, 1974; Welner *et al*, 1977, 1979). In brief, Bleuler believed that schizophrenia was a 'basic level' psychiatric category and that the presence of distinctively schizophrenic symptoms – regardless of the number or degree of affective symptoms – indicated its presence (Bleuler, 1911, 1924). Other researchers, rather than seeing schizoaffective disorder as a subtype of schizophrenia, have suggested that it is a subtype of affective disorder (Ollerenshaw, 1973; Pope & Lipinski, 1978; Baron *et al*, 1982; Goodnick & Meltzer, 1984) or have considered it to be a genetically independent, homogeneous entity (Mitsuda, 1967; Goode & Manning, 1983; Grossman *et al*, 1984; Harrow & Grossman, 1984).

However, not all theorists agree that schizoaffective disorder is either a subtype of one of the major psychoses or a homogeneous entity. Tsuang & Simpson (1984) and Levitt & Tsuang (1988) have proposed that schizoaffective disorder is, in fact, a genetically heterogeneous group which can be further subtyped into more homogeneous subgroupings. Yet another approach rejects the categorical model of psychiatric nosology altogether, and takes schizophrenia and affective disorder to be the opposing poles which define a continuum of impairment, with schizoaffective disorder falling somewhere in between (Beck, 1967; Crow, 1986). According to this view, schizoaffective disorder is a heterogeneous grouping of cases, some with more schizophrenic features and others with more affective features.

Samson *et al* (1988), in a review of studies which compared patients on the basis of global outcome measures, found that schizophrenic patients invariably had poorer outcomes and affective patients had better outcomes than patients with schizoaffective disorder (Post, 1971; Brockington *et al*, 1980*a*,*b*; Pope *et al*, 1980; Grossman *et al*, 1984; Maj, 1985). On the other hand, genetic findings, such as concordance rates of monozygotic and dizygotic twins for schizoaffective disorder, vary markedly depending upon the diagnostic criteria employed, the patients selected, and the terms used (Zerbin-Rüdin, 1986; Gross *et al*, 1986). Even though it is generally accepted that the relatives of schizoaffectives are at greater risk for psychiatric disorder in general, it is not at all clear whether they are at greater risk for particular disorders (Zerbin-Rüdin, 1986). Finally, in terms of family studies, schizoaffective disorder is generally seen as

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aetiologically and clinically heterogeneous (Rogers & Winokur, 1988).

Given this welter of complexity and contradiction in the literature, it is not surprising that there are no generally accepted criteria for schizoaffective disorder. Therefore, it is simply not possible to take current criteria and 'pick' a group of schizoaffective patients for study – the results would be confusing and unreliable. This being the case, we began with a population of atypical psychotic patients and gradually, through subtyping, narrowed our focus until we were left with a smaller group which could not be classified as either schizophrenic or affective. It is this more circumscribed group, defined as 'schizoaffective', which truly merits our scrutiny.

Method

In 1972, using strict diagnostic criteria (Feighner *et al*, 1972; Morrison *et al*, 1972), 200 schizophrenics, 100 patients with mania, and 225 patients with unipolar depression were selected for a study on major psychoses. These patients were chosen from among the 3800 total admissions to the Iowa State Psychopathic Hospital from 1934 to 1944.

Of the 510 patients admitted consecutively and clinically diagnosed as schizophrenic over the ten years, 310 failed to meet the Feighner criteria for schizophrenia. Patients were excluded for a variety of reasons: (a) schizophrenia was believed to be present but symptoms were not severe enough or of long enough duration to warrant a diagnosis; (b) two or more disorders were suggested by a mix of inconclusive symptoms (in particular, schizophrenia and affective disorder), or the symptom presentation was unusual or perplexing; (c) the patient's history lacked sufficient important information to make a definitive diagnosis; and (d) the patient suffered from alternating schizophrenic and affective episodes. We refer to these patients as the 'atypical psychoses' group.

From 1975 to 1987 follow-up information was collected on all probands and their first-degree relatives. Psychiatric diagnoses of first-degree relatives were made using a structured interview designed for this series of studies. The development of the interview form along with validity and reliability studies have been presented elsewhere (Tsuang *et al*, 1980*a*). Since the interviews were conducted 30–40 years after the proband's admission and the risk periods for schizophrenia and affective disorder are approximately between the ages of 15–39 years and 15–59 years respectively, most relatives were well beyond these risk periods. Hence, we were able to arrive at quite accurate measures of morbidity risks. If a relative was deceased and had been a patient at a psychiatric hospital, a diagnosis was made on the basis of the hospital record. Systematic psychiatric diagnoses of first-degree relatives, using all available clinical and demographic information, were made by psychiatrists who were blind to the research diagnoses of the probands and the probands' relations to family members. Final psychiatric diagnoses were based on a consensus among two or more psychiatrists.

Table 1
Description of first-degree relatives of those with schizophrenia, affective disorder and atypical psychosis

	Diagnostic group		
	Schizophrenia	Affective disorder	Atypical psychosis
Total no. of relatives	980	1974	2368
No. traced (% total)	895 (91)	1803 (91)	2320 (98)
No living (% traced)	484 (54)	885 (49)	1132 (49)
No. personally interviewed (% living)	354 (73)	683 (77)	701 (62)
No. of mental health records for deceased	21	47	73
Total interviews	375	730	774

The follow-up status of first-degree relatives of the schizophrenic, affective and atypical psychoses is presented in Table 1. First-degree relatives were traced to their current addresses if alive and, if deceased, death certificates or mental health hospital records were obtained. Between 91% and 98% of all such relatives were traced in this way and, in total, the number of interviews with them were: 375 for the schizophrenia group, 730 for the affective disorder group, and 774 for the atypical psychosis group.

Subtyping atypical psychoses

As noted, patients with 'atypical psychosis' are considered atypical precisely because, for any number of reasons, they fail to meet current diagnostic criteria for either schizophrenia or affective disorder. That is, the category 'atypical psychosis' is composed of a heterogeneous mix of patients whose one apparent common denominator is that they have all been excluded from other categories. A basic assumption of this study is that patients in the atypical psychosis group can be subtyped into more homogeneous subgroups and that ultimately it may be possible to pinpoint a schizoaffective group.

Our subtyping analysis began with the fact that statistically significant differences had previously been demonstrated between our typical schizophrenic and affective groups in terms of long-term outcome and familial data (Tsuang *et al*, 1979, 1980*b*). Invariably, the schizophrenics had significantly worse outcomes than the affective patients, and the morbidity risk for schizophrenia among the relatives of schizophrenics was significantly greater than for relatives of manics and depressives. Conversely, the morbidity risk of affective disorder was significantly greater in relatives of manics and depressives than in relatives of schizophrenics. In addition, based on long-term outcome, the atypical psychosis group fell between the schizophrenic and affective groups, with the atypical group having better outcome than the schizophrenics and poorer outcome than the affectives (Tsuang & Fleming, 1986).

With the above differences in mind we first needed to determine which variables significantly separated our sample of 310 atypical patients into definable subgroups. All patients (310 atypical patients, 200 schizophrenics, and 325 with an affective disorder) were systematically rated according to 50 demographic and symptom variables coded from the original hospital charts. Based on our previous work (Tsuang, 1979), we hypothesised that one of the subtypes would have predominantly schizophrenic features and one would have predominantly affective features. Hence, the demographic and clinical variables selected for the analysis needed to maximally distinguish schizophrenia from affective disorder at the 0.05 level. A second selection criterion was that the variables had to have an adequate distribution of values within the atypical group for the purposes of discrimination, with few missing values. Eleven variables that met the above criteria were: age at admission, sex, marital status, education, precipitating factors, self-reproach, poor premorbid adjustment, persecutory delusions, auditory hallucinations, hebe-catatonic motor symptoms, and short-term outcome.

The statistical technique used to subtype the atypical psychosis group was logistic regression (Feinberg, 1981; Engelman, 1981). This is a form of regression analysis which generates a prediction equation using a binary dependent variable and independent variables which may be either categorical or continuous. The dependent variable was 'diagnostic group' and was coded 1 for 'schizophrenia' and 0 for 'not-schizophrenia' (i.e. affective disorder). The independent variables were the 11 listed above. Age at admission is a continuous variable and the remaining ten variables are categorical, with (0,1) codings. The resulting regression equation, which predicted the probability of a subject being schizophrenic ($P(S)$), was then used on the complete atypical sample. Of the 310 patients in the atypical group, some who had missing data on one or more of the 11 predictor variables were omitted from the analysis, leaving 271 cases.

More details of the statistical method and the profiles of two patients, one who would have a high probability of being schizophrenic and the other who would have a low probability, are presented in the Appendix. The actual calculated probability values for being schizophrenic for each case were ordered from smallest ($P=0.2$) to largest ($P=0.97$) and their distribution is shown in Fig. 1. In order to determine if this was a continuous distribution we tested for normality. A Kolomogorov D statistic (Stephens, 1974) was calculated which rejected the hypothesis of normality. With this in mind we examined various cut-off points along the distribution of probabilities. By comparing different subdivisions of the distribution across the 11 variables, we determined that statistical breaks occurred at probabilities ($P(S)$) of 0.5 and 0.75. We suggest that these subdivisions, defined by the above cut-off points, represent three subtypes of the atypical group: (a) schizophrenic, ($n=111$, $P>0.75$); (b) undifferentiated ($n=57$, $P(S)=0.5$–0.75); and (c) affective ($n=103$, $P(S)<0.5$).

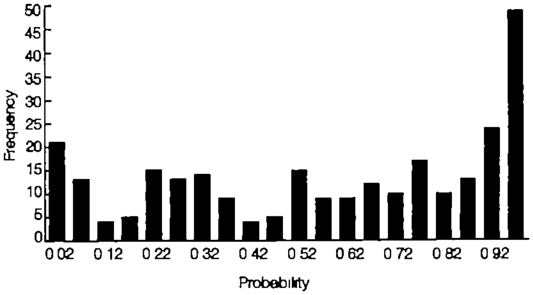

Fig. 1. Frequency distribution of probabilities for being schizophrenic (statistical breaks at $P=0.5$ and $P=0.75$ gives three subdivisions; $n=103$ ($P<0.5$), $n=57$ ($0.5<P<0.75$) and $n=111$ ($P>0.75$)).

Next, the 11 predictor variables and long-term outcome were compared across the three subtypes, and between the subtypes and the typical psychoses groups. These results indicated that the schizophrenic subtype was similar to the schizophrenia group and the affective subtype was similar to the affective disorders group. Results from the undifferentiated subtype on the other hand, were neither similar to the typical schizophrenia group nor similar to the typical affective disorder group. For this reason we considered the undifferentiated subtype to be 'schizo-affective disorder'. In order to examine this group further we analysed the morbidity risks of schizophrenia and affective disorder in the first-degree relatives. Differences in family data would be meaningful in helping to further define this group of schizoaffectives since the family information was not used in the subtyping selection process.

Results

Table 2 compares the morbidity risks for schizophrenia and affective disorder in the relatives of the schizoaffective, schizophrenic and affective groups. For schizophrenia, the relatives of schizoaffective patients had a morbidity risk (6.6%) that was comparable with that of the relatives of schizophrenics (5.5%), and significantly higher than that

Table 2
Morbidity risk of schizophrenia and affective disorder in relatives of those with schizoaffective disorder compared with relatives of those with schizophrenia and affective disorder

Proband group	n	Age-adjusted no. of relatives (BZ)[1]	No. of relatives with schizophrenia	Morbidity risk (%)	Age-adjusted no. of relatives (BZ)[1]	No. of relatives with affective disorder	Morbidity risk (%)
Schizoaffective	164	152.5	10	6.6	123.5	16	13.0
Schizophrenic	375	362.0	20	5.5	286.5	29	10.1
Affective	730	679.0	15	2.2*	531.0	96	18.1

*$P<0.05$ (comparison with schizoaffective disorder)

[1] BZ (*Bezugsziffer*) indicates age-adjusted lifetime risk period of ages between 15 and 39 and 15 and 59 years for schizophrenia and affective disorder respectively.

of relatives of affective patients (2.2%). For affective disorder, the morbidity risk for relatives of the schizoaffective group (13.0%) was lower than that found in relatives of the affective group (18.1%), and higher than that found in relatives of the schizophrenic group (10.1%) although no statistical differences were found.

The results support the view that the 'schizoaffective' group is neither a variant of schizophrenia nor a variant of affective disorder. On the one hand, for the risk of schizophrenia in relatives, the schizoaffective group is similar to the schizophrenia group and has a significantly higher rate of schizophrenia in their relatives than is found in the relatives of the affective disorders group. On the other hand, for the risk of affective disorder in relatives, the schizoaffective group is not significantly different from the affective disorders group and has a higher rate of affective disorder in their relatives than is found in the relatives of the schizophrenia group. This mix of morbidity risks suggests that schizoaffective disorder, as defined here, is different from schizophrenia and affective disorder.

Discussion

From a group of 310 atypical psychosis patients, we selected 57 cases which we take to be instances of schizoaffective disorder. In addition to these patients differing from patients in our typical schizophrenia and affective disorder groups on the basis of admission variables and long-term outcome, they also differed on the basis of family data. We compared the morbidity risks for schizophrenia and affective disorder in the relatives of the schizoaffective disorder group with such risks in the relatives of those with the typical psychoses. The results show that the schizoaffective patients, as defined here, are distinct from both schizophrenics and those with affective disorder.

In order to replicate these findings we are currently engaged in a follow-up and family study of atypical patients as defined by DSM-III-R (American Psychiatric Association, 1987). Our goal is to have samples of 200 schizophrenics, 200 bipolar disorders, 200 patients with major depression and 400 atypical psychosis patients in order to examine further those psychoses which differ from schizophrenia and affective disorder. Out of this, we hope to substantiate a new group of schizoaffective disorder cases based on the analysis of baseline, course and outcome, and family data.

In addition to schizoaffective disorder being intrinsically interesting as a subject for further research, its real importance lies in its relation to the major psychoses – as the boundary between the major psychoses. For example, using a dimensional model of psychiatric classification, the distribution of endogenous psychoses can be seen as a continuum, with schizophrenia at one extreme, affective disorder at the other extreme, and, if we imagine this to be a normal distribution, the majority of cases falling somewhere in between. Consequently, if schizoaffective disorder is broadly and loosely defined, it will encompass virtually the entire continuum, becoming diagnostically indistinguishable from 'endogenous psychosis', and hence meaningless, while diagnoses of schizophrenia and affective diagnosis will become unrealistically restrictive.

Alternatively, if we adhere to the traditional categorical model of psychiatric classification, schizoaffective disorder, as a category, still requires narrowly defined diagnostic criteria which can clearly distinguish it from schizophrenia on the one hand, and affective psychoses on the other hand. That is, if we develop narrow criteria to define the condition or category between schizophrenia and affective disorder, 'schizoaffective disorder' will refer to only a small band of 'fuzzy cases' between the major psychoses.

Hence, concerted effort should be made by researchers and clinicians alike to diagnose all possible cases of schizophrenia and affective disorder and leave only those cases which are truly 'different' to be classified as schizoaffective. Then rigorous, multimethodological research on schizoaffective disorder can be conducted by researchers using various theoretical models of the major psychoses, with some hope for real empirical and clinical convergence. Not only will this increase our understanding of schizoaffective disorder, but it will also heighten the critical contrasts between schizophrenia and affective psychosis.

Appendix

Statistical methodology and examples of calculating the probability of being schizophrenic

A version of standard logistic regression (Feinberg, 1981; Engelman, 1981) was used to calculate the probability for each of our atypical cases being schizophrenic ($P(S)$). In this analysis the dependent variable was binary, whereas the independent variables could be either categorical or continuous. We defined the dependent variable as $1 =$ schizophrenia and $0 =$ not schizophrenia. Therefore we rediagnosed our Feighner-criteria schizophrenics and affective disorder patients according to the more current DSM-III criteria, and used these cases to define the dependent variable. We wanted the statistical 'anchor points' ('schizophrenia', 'not schizophrenia') to be based on all information collected during our follow-up; this would allow for a more precise division of the atypical group into schizophrenic and affective subtypes. Eleven demographic and clinical factors coded from data collected at the time of admission were selected as independent variables. Based on the values of the independent variables

Table 3
Calculation of a high and low probability of being schizophrenic

Variable		High probability				Low probability		
		Design value	× Regression coefficient	Weight value		Design value	× Regression coefficient	Weight value
Age at admission: years		26	−0.076	−1.960		32	−0.076	−2.400
Sex	Male	−1	0.224	−0.224	Female	1	0 224	0.224
Marital status	Single	1	0.269	0 269	Married	−1	0 269	−0.269
Education	Secondary	−1	0.032	−0.032	Beyond secondary	1	0.032	0.032
Precipitating factors	None	−1	−0.326	0.326	Positive	1	−0.326	−0.326
Self-reproach	No	−1	−0.868	0.868	Yes	1	−0.868	−0.868
Poor pre-morbid adjustment	Yes	1	1.389	1.389	No	−1	1.389	−1 389
Persecutory delusions	Yes	1	0 563	0.563	Yes	1	0.563	0.563
Auditory hallucinations	Yes	1	0.630	0.630	No	−1	0.630	−0.630
Motor symptoms	Yes	1	0.439	0.439	No	−1	0.439	−0.439
Short-term outcome	Not recovered	1	1.331	1.331	Recovered	−1	1.331	−1.331
	Constant			2.148	Constant			2.148
			Linear function (u)	=5 767			Linear function	=−4 685

$$\text{Probability of schizophrenia} = \frac{e^{u}}{1+e^{u}} = \frac{e^{5\,767}}{1+e^{5\,767}} = 316.4/317.4 = 0.99$$

$$\text{Probability of schizophrenia} = \frac{e^{u}}{1+e^{u}} = \frac{e^{-4\,685}}{1+e^{-4\,685}} = 0.009/1\,009 = 0.009$$

and the computer-generated regression coefficients, the probability of a patient being schizophrenic ($P(S)$) was calculated as follows:

$$P(S) = \frac{e^{B1+B2(t1)+B3(t2)+}}{1+e^{B1+B2(t1)+B3(t2)+}}$$

This formula represents the logistic model where B1 is a constant, B2, B3. . . are regression coefficients, t1, t2. . . are computer-generated design values, and 'e' represents the exponential function. For our categorical independent variables, design values are −1 for variables coded as zero, and 1 for variables coded as one. For the continuous independent variable (age at admission) the design value is the actual coded value.

To illustrate this method, two hypothetical examples are given: a patient who has a very high probability of being schizophrenic and one who has a very low probability of being schizophrenic (Table 3). As shown in Table 3, the design values for each of the ten categorical variables are multiplied by the regression coefficients to obtain weighted values. For age at admission, which is a continuous variable, the regression coefficient is multiplied by the actual age. These weighted values and the constant (B1) are summed to form a linear function, shown as (u) in Table 3. The linear function (u) is inserted in the exponential formula which is evaluated to obtain $P(S)$. Thus, in the first example $P(S)=0.99$ compared with $P(S)=0.009$ in the second example. For each of our 271 patients with atypical psychoses we applied the above formula using the actual demographic and clinical information collected at index admission to the psychiatric hospital.

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Pre-morbid Adjustment and Personality in Psychosis
Effects of Sex and Diagnosis

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Pre-morbid schizoid and schizotypal traits and social adjustment were assessed blind to diagnosis by interviewing the mothers of 73 consecutively admitted patients with DSM-III schizophrenia or affective psychosis. Analysis of factors associated with pre-morbid deficits showed a highly significant interaction of diagnosis with sex, such that schizophrenic men showed much greater pre-morbid impairment than either schizophrenic women or men with affective disorder. Poor pre-morbid adjustment predicted an early age at first admission The results can be explained by a neurodevelopmental disorder in some schizophrenic males.

Some adults with schizophrenia were odd as children. The traits most commonly noted involve those deficits of cognition and affect which together comprise Kretschmer's (1921) description of schizoid personality: solitariness, cold affect and eccentricity. Other traits which are often cited are suspiciousness, rigidity and unusual speech. These observations date back to Kraepelin and Bleuler, yet the nature of the relationship between the pre-morbid abnormalities and adult schizophrenia remains obscure. Are they precursors of schizophrenia specifically or of severe mental illness in general? What proportion of schizophrenic patients had such traits as children?

In his review of several early German studies which examined the apparent prevalence of abnormal pre-morbid traits in schizophrenia, Cutting (1985) concluded that overall about a quarter of patients were considered schizoid pre-morbidly, and that a further one-sixth were abnormal in other personality traits. The nature of the link between these abnormal personality traits and the emergent psychosis has been explained in several different ways. The psychotic symptoms have been seen as a reaction to stress in a peculiarly vulnerable personality (Kretschmer, 1921), as a fundamental part of the extended phenotype of schizophrenia (Meehl, 1962), or simply as the early stage of a longitudinal disease process (Kraepelin, 1913). 'High risk', twin and adoption studies (Kendler *et al*, 1981; Mednick *et al*, 1987) have clarified the genetic relationship between abnormal personality and schizophrenia and have enabled better definition of the traits involved with the operationalised concept of schizotypal personality (American Psychiatric Association, 1980). Studies looking back at pre-morbid personality in cohorts of schizophrenic patients are prone to the difficulties involved in retrospective interpretation. Nonetheless, they remain important in view of the impact that abnormal pre-morbid personality appears to have on the nature of the subsequent illness. It seems to predict early onset and poor prognosis (Strauss *et al*, 1977), cognitive deficits and negative symptoms, as well as correlating with cerebral ventricular size in some reports (Weinberger *et al*, 1980; Williams *et al*, 1985).

These observations have given rise to neurodevelopmental formulations of schizophrenia. Broadly, these propose the existence of a non-progressive brain lesion of genetic or early environmental origin. The cognitive and behavioural effects of such a lesion are postulated to change over time as the nervous system around it continues to develop. In the immature brain, the functional effects are subtle, with relatively minor deficits in trait characteristics such as affective responsiveness and sociality. Only as the brain reaches functional maturity in adolescence do the psychotic symptoms of delusions, hallucinations and thought disorder become manifest (Weinberger, 1987; Murray & Lewis, 1987; Lewis, 1989).

The current study investigates pre-morbid personality and adjustment in an operationally defined sample of young psychotic patients. In order to standardise collection of information and maximise its validity, the mother was the selected informant in each case. Accordingly, the sample comprised a consecutive series of hospital admissions for whom the mother was available for interview. In all cases, mothers were interviewed by a rater blind to the diagnostic status of the patient. The main hypothesis was that the frequency and severity of pre-morbid deficits would be increased in patients with schizophrenia compared with those with affective psychosis. In the light of recent interest in the effects of gender on age at onset, course and prognosis of schizophrenia (Goldstein, 1988; Angermeyer *et al*, 1989), this was planned as the second independent variable of interest.

Our predictions were that pre-morbid personality and adjustment deficits:

(a) characterise schizophrenia rather than affective psychosis
(b) are found in men more than in women
(c) predict early onset of psychotic symptoms in schizophrenia.

Method

A total of 1039 consecutive admissions over 12 months to acute general psychiatric wards of the Royal Bethlem and Maudsley Hospital were screened for symptoms of functional psychosis. Operational criteria for functional psychosis were defined as the presence of delusions, hallucinations or thought disorder during the current episode in the absence of clinically manifest cognitive impairment. Patients aged 16–50 years were included if, at interview, they: (a) had a DSM–III diagnosis of schizophrenia (295.1, −2, −3, −9) or affective disorder with psychotic features (296.24, −34, −44, −54, −64) (American Psychiatric Association, 1980); (b) had a mother available for interview (i.e. living in UK or Eire); and (c) had psychotic symptoms not secondary to effects of drugs or alcohol.

Although 94 patients met these criteria, in 10 cases the patient refused access to the mother and in 11 cases the mother herself refused an interview. Thus, a final sample of 73 patients who fulfilled these criteria had prospective diagnostic interviews and separate, blind maternal interviews for pre-morbid data.

The instruments used were developed for this study and included an assessment of pre-morbid and schizotypal traits (PSST) and an assessment of pre-morbid social adjustment (PSA). The former interview was developed to cover areas of long-standing pre-morbid dysfunction relevant to current concepts of schizoid and schizotypal disorders. Seven items were scored for the period between the ages of 5 and 16 years, each with a standardised entry question and rates on a four-point (0–3) scale with explicit anchor points (3 = most impaired). The items addressed sociability ('active social interaction' to 'withdrawn and isolated with no friends'); demonstrative affect ('warm with spontaneous shows of affection' to 'cold and aloof with all'); suspiciousness/sensitivity ('never unduly suspicious or socially anxious' to 'marked social anxiety, suspiciousness, distrustfulness plus paranoid ideation'); ideas of reference/perceptual distortions ('none' to 'daily expression of odd ideas/unusual perceptions/ideas of reference'): speech ('normal' to 'consistent pattern of clearly deviant speech, with examples'); socialised and unsocialised antisocial behaviour ('never' to 'severe and repeated').

Mothers were instructed to give full answers and to consider enduring personality traits before the first episode of psychosis. Seven of the eight schizotypal, and three of the four schizoid, features listed in DSM–III were included. Copies are available from the authors.

The PSA was derived from the Cannon-Spoor scale for pre-morbid adjustment (Cannon-Spoor *et al*, 1982), adapted for a UK sample. Thus, five items were included, each with an eight-point rating scale with explicit anchor points. The items covered socialisation, peer relationships, scholastic performance, school adaptation, and hobbies and interests. Each item was rated for two periods roughly equivalent to time at primary school and time at secondary school: 5–11 years (PSA1) and 12–16 years (PSA2). Two cases had been admitted as in-patients for psychosis by age 15 years: in each case, PSA1 was rated, but not PSA2 nor PSST.

Inter-rater reliability for these scales was assessed by a second rater (SL) independently rating a random sample of eight consecutive audiotaped interviews (AF). Intraclass correlation coefficients were calculated which showed reliability to be satisfactory for PSA1 ($r = 0.64$; $P < 0.05$) and PSA2 ($r = 0.64$; $P < 0.05$), and good for PSST ($r = 0.98$; $P < 0.001$).

Procedure

Mothers were interviewed by a rater blind to diagnostic information on the proband. A total of 30% of the maternal interviews were conducted by telephone.

Patients were interviewed by a separate rater blind to maternal information. Symptom assessment was by PSE–9 (Wing *et al*, 1974), which was used in addition to clinical and case-note information and adapted to yield DSM–III axis-1 diagnoses. The interview was conducted within 14 days of in-patient admission. Age at first hospital admission for psychotic symptoms was recorded: this index was chosen rather than age of onset in view of its high reliability and compatability with other studies (Hafner *et al*, 1989).

Results

Seventy-three mothers of psychotic patients were interviewed. Details of the 73 patients are given in Table 1. Three pre-morbid ratings were obtained from the maternal informant: pre-morbid schizoid and schizotypal traits (PSST), pre-morbid social adjustment at 5–11 years (PSA1) and pre-morbid social adjustment at 12–16 years (PSA2). PSST was rated in 61 cases, PSA1 in 67 cases and PSA2 in 63 cases: the numerical discrepancies arose because a rating of PSST required the patient to have been in contact with the mother up until 16 years, while PSA1 and PSA2 required contact at 5–11 and 12–16 years respectively. Separations due to emigration, partial rearing by third

Table 1
Details of patients ($n = 73$)

	DSM–III diagnosis	
	Schizophrenia ($n = 45$)	Unipolar/bipolar disorder with psychotic features ($n = 28$)
Mean age at assessment (s.d.): years	26.8 (5.6)	30.1 (8.2)
Ratio male:female	35:10	12:16
Mean age at first in-patient admission (s.d.): years	22.6 (5.3)	24.9 (7.5)

parties and first admission before age 16 years precluded full ratings in some eligible patients.

Not surprisingly, correlations between the three rating scores were significant. Between PSA1 and PSA2, Spearman's correlation was 0.90; between PSST and PSA1 it was 0.45; between PSST and PSA2 it was 0.49 ($P<0.001$ in each case).

Of the 61 patients rated for PSST, 37 had a diagnosis of schizophrenia compared with 24 with a diagnosis of bipolar or unipolar affective disorder (with psychotic features).

Effect of diagnosis and sex

PSST was scored on a range 0–21. Of the 61 rated, 47 (61%) scored less than 2; the score distribution had a marked positive skew. Similar distributions were noted for PSA1 and PSA2. The rating scores were therefore log-transformed and entered into an analysis of variance with DSM-III diagnosis and sex. For PSST, a significant main effect was seen for diagnosis ($F=6.7$, $P=0.01$); a trend only was seen for sex ($F=3.6$, $P=0.07$). Thus, schizophrenic patients' mothers reported significantly more schizoid and schizotypal traits than did the mothers of affective patients; there was a trend towards more such traits in men.

The analysis of variance showed a significant two-way interaction between diagnosis and sex on PSST score ($F=10.6$, $P=0.002$). Thus, male schizophrenics were rated much more highly for PSST than other groups.

Similar findings emerged for PSA2 with a significant main effect for diagnosis ($F=4.7$, $P=0.03$) but not sex; again, the two variables showed a significant two-way interaction ($F=4.5$, $P=0.03$). PSA1 showed no significant main or interaction effects for diagnosis and sex.

Mean PSST scores were similar in men and women with affective disorder and in schizophrenic women (7.40; 7.86, 7.17), whereas the mean PSST score for schizophrenic men was 9.61. Schizophrenic men had more pre-morbid schizoid and schizotypal features than both schizophrenic women (Mann–Whitney U, $Z=3.0$, $P<0.001$ one-tailed) and men with affective disorder ($Z=3.3$, $P<0.0005$) (Table 2). Mean PSA2 scores showed a similar pattern, with men and women with affective disorder and schizophrenic women scoring 9.27, 10.69 and 10.14 respectively, compared with a mean of 14.00 for schizophrenic men. Schizophrenic men had a significantly higher PSA2 score than schizophrenic women ($Z=1.7$, $P<0.05$) or men with affective disorder ($Z=2.5$, $P=0.005$). Thus, the pre-morbid impairments were neither a function of sex alone, nor diagnosis alone: only being male and pre-schizophrenic predicted impairments

Table 2

Mean scores of pre-morbid schizoid-schizotypal traits (PSST) and early and late childhood social adjustment (PSA1, PSA2) in men and women with schizophrenia or affective psychosis

	Schizophrenics	Those with affective psychosis
Men		
mean PSST	**9.61***[1]	7 40
mean PSA1	12 83*	9 50
mean PSA2	*14 00*	9.27
Women		
mean PSST	7.17	7.86
mean PSA1	11.00	9 87
mean PSA2	10.14	10 69

*$P<0\ 05$, **$P<0\ 005$, ***$P<0\ 0005$

[1] Planned univariate, non-parametric comparisons (Mann–Whitney, one-tailed) left-hand asterisks relate to comparisons between men and women with schizophrenia, right-hand asterisks relate to comparisons between men with schizophrenia and men with affective psychosis

Age at first admission

For the group as a whole, age at first in-patient admission correlated negatively with all pre-morbid measures: PSST $r=-0.18$, $P=0.08$; PSA1 $r=-0.31$, $P=0.005$; PSA2 $r=-0.23$, $P=0.03$ (Spearman's r) It is notable that PSA1 showed the highest correlation with early age at admission This suggests that the marked pre-morbid deficits shown above in schizophrenic men are unlikely to be due to an early, gradual onset of adult psychosis being confused with pre-morbid deficits, since measures before age 11 were actually the best predictors of early onset. Admission age rather than estimated age at 'onset' was used also to circumvent this problem, as was the exclusion of those in whom first admission was before 16 years of age.

An unexpected finding was that the measures of social adjustment, PSA1 and PSA2 scores, were stronger predictors of early admission in affective disorder ($r=0.52$, $P=0.004$; $r=-0.44$, $P=0.001$) than they were in schizophrenia ($r=-0.22$, NS; $r=0.10$, NS). This might be a type-II statistical error resulting from the under 16 exclusion rule making for a narrow range of first admission ages in the schizophrenic group. Nonetheless, it is an interesting result which appears to demonstrate the importance of pre-morbid factors in determining age at presentation of adult affective psychosis. PSST score was more strongly correlated with early onset in schizophrenia ($r=-0.20$) than in affective disorder ($r=-0.09$), although in neither group was this correlation significant.

Could the correlation between pre-morbid deficits and early age at admission in the whole group explain the observed pre-morbid differences between the schizophrenic and affective disorder groups, since age of onset for schizophrenia is younger in general than for affective disorder? Age at first admission was entered as a covariate in the foregoing ANOVA calculations to test this For PSST, entering age at first admission did not affect the ANOVA results greatly. although a significant covariate ($F=4.7$, $P=0\ 004$), it altered the main effect of diagnosis only slightly ($F=6.3$, $P=0.02$) and actually strengthened the two-way interaction between sex and diagnosis ($F=12.3$, $P=0.001$). Neither did entering age as covariate materially alter the results reported above for PSA1 and PSA2. As with PSST, the sex–diagnosis interaction for PSA2 actually increased when age at admission was taken into account ($F = 5.6$, $P = 0.002$). Thus, differences in age at first admission (or age at 'onset') did not account for the two diagnostic groups' differences in pre-morbid factors. Indeed, age at first admission was similar (Table 1) in the two groups.

Discussion

Over 12 months, we assessed 73 consecutive in-patient admissions aged 16–50 years who met DSM-III criteria for schizophrenia or affective disorder with psychotic features. In each case, standardised interviews with the patient's mother were conducted by a separate interviewer blind to patient diagnosis, to assess pre-morbid personality and adjustment. We found that abnormal pre-morbid personality with schizoid and schizotypal traits (PSST) was reported significantly more frequently in the schizophrenic patients than in those with affective psychosis. Moreover, schizophrenic men specifically were highly significantly more abnormal in pre-morbid personality than other groups, as shown by a two-way interaction effect between sex and diagnosis. Women with schizophrenia differed little from men and women with affective disorder on these pre-morbid measures. Similar effects were apparent with reported adjustment at secondary-school ages.

In the series as a whole, all poor pre-morbid personality adjustment measures predicted significantly earlier age at first psychiatric in-patient admission. Social adjustment at primary-school age was the strongest predictor. Unexpectedly, these correlations were stronger for affective disorder than for schizophrenia.

What do these results mean? First, it is important to note the main methodological limitation of the study: the retrospective assessment of pre-morbid characteristics. We endeavoured to eliminate likely biases in three ways. Information was collected from the biological mother in all cases and in each case only for those periods in which the mother was in close contact with the patient. A control group was chosen in which illness severity (and, fortuitously, age at first admission) in the acute phase was similar to that of the schizophrenic group: affective disorders with psychotic features. This minimises any possible artefactual effect of the illness severity influencing the mother's recall or report of abnormalities in childhood. Any possible interviewer bias was reduced by the use of an interviewer blind to clinical and diagnostic data regarding the patient. The face validity of the interview to collect pre-morbid data was thus increased, and inter-rater reliability was shown to be acceptable. The accuracy of the data ultimately remains unknown, but systematic bias between diagnostic groups was rendered unlikely.

The results confirmed our first prediction, that a diagnosis of schizophrenia was significantly associated with reported pre-morbid schizoid-schizotypal traits and late-childhood social impairments (PSA2). This supports the notion that such pre-morbid abnormalities are more characteristic of schizophrenia than of the other psychoses. Late childhood impairments in adjustment (PSA2) were more predictive of schizophrenia than were early impairments (PSA1), a finding which accords with evidence that, although schizoid-schizotypal traits can be detected in early childhood (Hanson *et al*, 1976; John *et al*, 1982), their impact on broader social adjustment emerges most strongly in later childhood (Watt, 1972). The high correlation noted between early childhood social adjustment (PSA1) and age at first admission to hospital for psychosis suggests that where deficits are severe enough to impair social adjustment at an early stage, these are powerful predictors of early onset. This finding was not diagnosis-specific and actually was more pronounced for affective psychoses, perhaps as a result of the greater variance in age at admission to hospital in this group.

Deficits were relatively specific to boys who were to develop schizophrenia, compared with girls who were to develop schizophrenia or boys who were to develop affective psychosis. The last two groups differed little from each other in measures of schizoid-schizotypal personality or social adjustment, or from the fourth group: girls who were to develop affective psychosis (Table 2). How can we explain this finding? Two possible artefacts should be first considered. It could conceivably be that the male schizophrenic group had a particularly early or insidious onset of psychosis, the initial features of which were interpreted by mothers as poor personality and adjustment. Inspection of the data analysis enables us to reject this explanation. The age at first admission to hospital was not significantly earlier in male schizophrenics than the other groups. Furthermore, age at first admission was controlled for in an analysis of covariance which strengthened rather than weakened the association. The second possible artefact is less easy to test: the traits of interest might be present in pre-schizophrenic girls but are not recognised as such by mothers because of some sex-related expectations of social behaviour. However, this would not explain the observed differences between the two male diagnostic groups. Nor would it fit with what is known of sex differences in childhood temperament and social behaviour: if anything, girls are more empathic and 'sociable' (Hoffman, 1977) than boys, especially in relation to their mother (Maccoby & Jacklin, 1980) and thus any deviance in their social development should be more, not less, easily detectable by mothers.

If the finding is real, then it has implications for research into the causes of schizophrenia. Diagnosis-dependent differences in pre-morbid characteristics have been well researched; the interacting effect with sex has not. Sex differences in certain aspects of the course of schizophrenia have been known for a considerable time. In particular, that median age of onset is earlier in men than in women is well established, although still unexplained (Lewine *et al*, 1981; Hafner *et al*, 1989). Prognosis also seems better in women (Loranger, 1984) while men are more likely to show defect-state symptoms (Goldstein & Link, 1988). Poorer pre-morbid adjustment in male compared with female schizophrenic patients has been noted (Kloreman *et al*, 1977; Zigler & Levine, 1984) but the specific interaction of sex and diagnosis was not examined in these studies although a review of the evidence for pre-morbid intellectual impairments in schizophrenia concluded that, where such deficits exist, they are mostly a male characteristic (Aylward *et al*, 1984). Conventional genetic hypotheses (Gottesman & Shields, 1982) would be hard-put to explain the apparent specificity of pre-morbid deficits to schizophrenic men. A polygenic-multifactorial model might invoke sex-dependent thresholds, but this would require that, if the male form of the disorder were more severe, it should also be considerably less prevalent; this is not the case. A single major locus model could explain the data only if an unusual sex-specific interaction at the cellular level were invoked, as in the recent model of pseudoautosomal X-linkage posited by Crow *et al* (1989). One attempt to explain sex-differences in onset proposed a hormonal protective factor in women (Seeman, 1983). Such a mechanism would not explain our findings of pre-pubertal differences, particularly in the absence of an observed effect concerning age at first admission to hospital.

A possible explanation for the findings is that there exists a subtype of schizophrenia which is characterised by particular deficits of pre-morbid functioning, and that this subtype is more commonly found in men. There exists a ubiquitous excess of males in recognised neurodevelopmental disorders such as epilepsy, stuttering and severe mental retardation. Recent evidence from brain imaging studies suggests that the non-progressive abnormalities seen in schizophrenia are also found predominantly in men (Andreasen *et al*, 1990). Childhood deviance in social adjustment and personality might therefore be an early behavioural manifestation of a neurodevelopmental subtype of schizophrenia which is most often found in males.

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Comparison of CATEGO-Derived ICD–8 and DSM–III Classifications using the Composite International Diagnostic Interview in Severely Ill Subjects

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Two classifications, DSM–III and CATEGO4-derived ICD–8, included in the CIDI, are compared in 63 in-patients and out-patients with a broad range of psychiatric illness. Agreement for main diagnosis between these classifications was statistically significant for two time frames, the present state and lifetime However, while diagnostic assignment by CATEGO4 remained fairly constant between time frames, there was a marked shift in DSM–III-assigned diagnosis, with cases changing from anxiety state diagnosis (present state) to affective disorder (lifetime). Thirty-nine subjects were assigned a DSM–III diagnosis of affective disorder for lifetime illness compared with 21 assigned to this diagnostic group by CATEGO4.

In this paper we compare two classifications of psychiatric disorder that can be derived from the Composite International Diagnostic Interview (CIDI; Robins *et al*, 1985) – DSM–III (American Psychiatric Association, 1980) and CATEGO(Wing *et al*, 1974; Wing & Sturt, 1978).

The CIDI has been developed from the Diagnostic Interview Schedule (DIS; Robins *et al*, 1981) for use in epidemiological research; the instrument's historical development and structure has been described in detail (Robins *et al*, 1988). Using a computer scoring program, diagnoses can be made according to DSM–III as well as other operational criteria (Feighner *et al*, 1972; Spitzer *et al*, 1975). In addition it is possible to hand-score CIDI. In developing CIDI from the DIS, the first 40 items and certain psychotic items from the Present State Examination (PSE; Wing *et al*, 1974; Robins *et al*, 1988) were included (although with different wording, scaling, and method of interview) so that certain CATEGO classes could be derived along with their tentative equivalents from ICD–8 (World Health Organization, 1974). In 1984 CIDI was approved by the World Health Organization (WHO) for international use in psychiatric research, and field trials of a "core version" of the instrument have recently been completed in 19 centres around the world.

Previously (Farmer *et al*, 1987 *a*), we evaluated the agreement between the PSE items in CIDI and the PSE given in separate interviews of the same subject. Although we found that item-by-item inter-rater agreement between the two interviews was variable and sometimes poor, when the PSE ratings from both interviews were scored by the CATEGO computer program, agreement for syndrome classification, diagnostic class and severity attained satisfactory statistical significance. In this study we assume that CATEGO scores from the CIDI approximate satisfactorily to those provided by the PSE and we therefore compare this classification with DSM–III.

The version of CATEGO that has been used to score the PSE items in CIDI is the fourth edition (CATEGO4). The CATEGO4 program groups together individual symptoms into 38 'syndromes'. These in turn are grouped into six subclasses. Finally, one CATEGO class is specified as the most likely diagnosis and a tentative ICD–8 category is suggested. This approach to classification is therefore somewhat different from the well known operational approach of DSM–III.

While the CATEGO4-derived ICD–8 classification may be considered more flexible, it provides less certainty of diagnosis and in particular gives weighting to certain symptoms or groups of symptoms. The program is probabilistic in its decision making. In contrast, operational criteria such as DSM–III produce greater certainty in assigning diagnosis but in general more cases fall outside the prescribed criteria into miscellaneous categories or categories of "not elsewhere specified".

In this paper comparisons are made across broad diagnostic groups only, for although highly comparable for three digits, ICD–8 and DSM–III deviate from each other at the four- and five-digit level. We report on the degree of agreement and disagreement between the two systems, and examine sources of disagreement to see if any trends emerge.

Method

Sixty-three subjects were chosen to represent a wide range of clinical diagnoses. Thirty subjects have been described previously (Farmer *et al*, 1987 *a*), including ten patients with serious psychotic illness (six women, four men), selected from a consecutive series of patients admitted with a clinical diagnosis of psychosis, to the psychiatric ward of a district general hospital. Ten of these 30 patients (four men, six women) constituted a consecutive series of non-psychotic out-patients, seen by one psychiatrist at the Department of Psychological Medicine, King's College Hospital, London, and ten subjects were out-patients attending the Eating Disorders Unit at the Maudsley Hospital, London, nine of whom were women suffering from eating disorders and one of whom was a man who had a primary clinical diagnosis of neurotic depression. Lastly, there were 33 subjects (20 women, 13 men) who were in-patients and day patients at the Acute Psychiatric Unit at the University Hospital of Wales, Cardiff, and Whitchurch Psychiatric Hospital, Cardiff.

All patients were interviewed using the CIDI by psychiatrists (AEF and PLJ) or psychologists (RK and LR) trained in its use. Ratings for PSE–9 items from CIDI were then transcribed to PSE–9 coding sheets for past-months and lifetime frames by two graduate students in clinical psychology who had no knowledge of PSE–9 or CATEGO4 but who were given explicit transcription rules. The codes were then entered into the computer program CATEGO4 to obtain PSE syndrome frequencies, CATEGO class, and suggested ICD–8 diagnosis.

Broad ICD–8 diagnostic groups are compared with corresponding DSM–III categories, namely: 300 (anxiety states, phobic disorders, obsessive–compulsive disorders, neurotic depression), 295 (schizophrenic psychoses), 296 (affective psychoses), 297 (other psychoses) and, lastly, no diagnosis. As category 300.4 denotes separate disorders for DSM–III and ICD–8, the classifications are also compared for PSE syndrome profiles for 300.4 as well as 296 depression categories.

The DSM–III diagnosis was obtained blind to CATEGO4 data using a hand-scoring method (AEF) as the full computerised scoring program for CIDI was still being developed. As most subjects fulfilled several operational definitions for the same time frame, a clinical decision based on reference to hospital case records was made by AEF regarding the principal axis 1 DSM–III diagnosis. Both the present-state and lifetime diagnoses for DSM–III were produced for comparison with CATEGO4. For both time frames, the principal axis 1 diagnosis was employed in this comparison exercise.

Results

The interviewed sample consisted of 22 men and 41 women, mean age 42 (range 17–81 years). The full table of diagnostic assignments for DSM–III and CATEGO4 for each subject may be obtained upon request from the first author.

In order to apply tests of association between DSM–III and CATEGO4-derived ICD–8 classifications we must commence with the assumption that these are derived from two independent measures (Cicchetti, 1978). In practice this is not the case, since our results are derived from a single interview of each subject. In order to test whether we can assume that diagnostic assignment is independent and not related to case identification, a hierarchical log-linear analysis was carried out (Frude, 1987) (Table 1). Case number (CN) and DSM–III and ICD–8 diagnoses were entered as factors, and only DSM–III and ICD–8 classification proved significant, there was no significant factor-by-factor interaction. Thus it is reasonable to conclude that association between DSM–III and ICD–8 classifications were independent of subject identity in the subsequent analyses.

Table 1
Hierarchical log-linear analysis with case number (CN) and DSM–III and CATEGO4 diagnoses entered as factors

	d f	Partial χ^2	Probability
CN × DSM–III	248	82.061	1.000
CN × CATEGO4	248	81.344	1.000
DSM–III × CATEGO4	16	0.000	1.000
CN	62	28.933	0.999
DSM–III	4	29.183	0.000
CATEGO4	4	29.900	0.000

Table 2 shows the cross-tabulations for DSM–III and ICD–8 present and lifetime diagnoses using the broad diagnostic groups described in the method. Chi-square and contingency coefficients for both time frames indicate associations which are comparatively strong and statistically significant (present state, $\chi^2=47.64$, d.f. = 9, $P=0.000$; lifetime, $\chi^2=60.35$, d.f = 9, $P=0.000$). In addition, diagnostic assignment for DSM–III and CATEGO4 for each subject for each time frame was entered into a computer program (Frude, 1987) to assess diagnostic agreement via the kappa statistic, specifying the data as nominal (Cohen, 1960). For present state, percentage observed agreement was 57%, percentage expected agreement 26%, kappa = 0.418, P of kappa = 0.00001; and for lifetime, percentage observed agreement = 59%, percentage expected agreement = 31%, kappa = 0.399, P of kappa = 0.00001. Thus, on different tests of statistical association, agreement for diagnostic assignment between the two classifications is significantly better than chance. However, conventionally the findings for the kappa statistic are regarded as showing poor agreement for diagnostic assignment.

From the cross-tabulations in Table 2, the following observations can be made. For present state, 14% and 16% of cases (DSM–III and ICD–8 respectively) are not assigned to any diagnostic group. DSM–III does not assign any cases to the 297 (other psychoses) group although four cases are assigned to this category by CATEGO4. Both classifications assign approximately 30% of cases to the 300 (anxiety states) group. The main difference between the two classifications for present state diagnoses is shown by the percentage of cases assigned to the 295 (schizophrenic psychoses) and the 296 (affective psychoses) categories. DSM–III assigns the majority of cases (43%) to the affective psychoses category but only 13% to the schizophrenia category. In contrast, CATEGO4 assigns 25% of cases to 296 and 27% of cases to 295.

Table 2

Cross-tabulations of DSM-III and ICD-8 diagnoses from CIDI and kappa values

DSM-III	ICD-8 present state						ICD-8 lifetime					
	no diagnoses	300	296[1]	295/297[2]	totals	kappa of agreement	no diagnoses	300	296	295/297[3]	totals	kappa of agreement
No diagnosis	5	4	0	0	9 (14%)	0.44	2	2	0	0	4 (6%)	0.65
300	4	9	3	3	19 (30%)	0.22	0	7	2	2	11 (17%)	0.29
296[1]	1	7	13	6	27 (43%)	0.21	0	11	19	9	39 (62%)	0.35
295	0	0	0	8	8 (13%)	0.56	0	0	0	9	9 (14%)	0.53
Totals	10 (16%)	20 (32%)	16 (25%)	17 (27%)			2 (3%)	20 (32%)	21 (33%)	20 (32%)		

1 This includes 300 4/296 2

2 Four cases assigned to 297 by CATEGO4 are included in the 295 group for the purpose of the analysis No cases assigned to 297 by DSM-III

3 Five cases assigned by CATEGO4 are included with 295 cases for the purposes of this analysis No cases assigned to 297 by DSM-III

For lifetime disorder (Table 2), fewer cases are assigned to the no-diagnosis group, which is to be expected since all subjects were receiving some type of psychiatric care at the time of interview. Thus subjects with only minor symptoms for the present state may have been under psychiatric review as they were still recovering from a severe episode of illness. However, 6% of cases for DSM-III and 3% of cases for CATEGO4 received no lifetime diagnosis. Once again, no cases were assigned to the 297 category by DSM-III while five cases were assigned to this group by the CATEGO4 program.

Fewer cases were assigned to the 300 (anxiety state) group for lifetime compared with present state by DSM-III. However, the same percentage (32%) were assigned to this group of diagnoses by CATEGO4. Once again, the main difference between the two classifications is found in the assignment to the 295 and 296 groups. Of DSM-III cases 62% were assigned to 296, while only 33% were by the CATEGO4 classification. Conversely, 14% of DSM-III cases were assigned to 295 compared with 32% categorised as schizophrenic psychoses according to CATEGO4.

It can also be seen that the broad diagnostic agreement for current illness for DSM-III and CATEGO4 is 56% and for lifetime illness is 59%. Conversely, there is 44% (28 cases) disagreement over present state, and 41% (26 cases) disagreement over lifetime illness. Three main sources of disagreement are examined: firstly, cases that are on the borderline of illness, i.e. where one classification assigns a category (usually 300 or 296) while the other classification fails to assign a diagnosis. Nine cases (32% of disagreements) are on the borderline between illness and not ill for present state and two cases (7%) for lifetime.

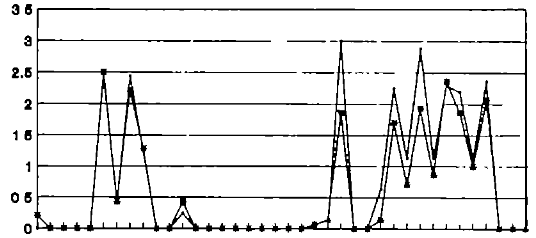

Fig 1 The mean frequencies of 38 PSE syndromes for 296 2 and 296.3 categories ($n = 63$) (ICD-8, —■— (296.2 only), DSM-III, —+—(296.2 plus 296.3)).

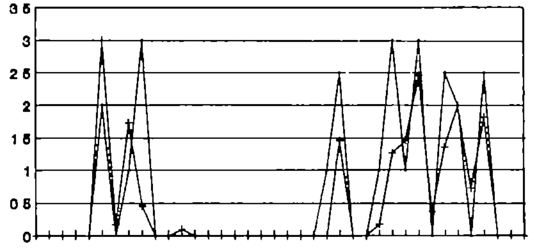

Fig. 2 The mean frequencies of 38 PSE syndromes for the 300.4 category ($n = 63$) (ICD-8, +, DSM-III, —■—)

The second source of disagreement is between a psychosis (295, 296 or 297 category) and a non-psychotic disorder (300 category). Here there is disagreement in 13 cases (46%) for present state and 15 cases (58%) for lifetime. Lastly, there is disagreement concerning type of psychotic illness, where one classification assigns a 296 (affective disorder) category while the other assigns to 295 or 297 (schizophrenic or other psychoses). Disagreement for present state occurred in six cases (21%) and for lifetime in nine cases (35%) As we have already noted, there is a marked tendency for DSM-III to assign to affective disorder subjects classed as schizophrenic by the CATEGO4 program.

The mean frequencies for 38 syndromes for 296.2, 296.3 and 300.4 categories are shown in Figs 1 (296) and 2 (300.4). CATEGO4 assignment for 11 cases was neurotic depression (N+; ICD-8 300.4) and for 17 cases was retarded depression (R+; ICD-8 296.2 or 300.4). Principal Axis 1 DSM-III diagnosis for two subjects was 300.4 and for 14 subjects was 296.22 or 296.32.

Discussion

While there have been previous comparisons of different classifications, none to our knowledge have used a single interview to derive two rather different nosologies such as DSM-III and CATEGO-derived ICD-8. Clearly there are a number of caveats which should be addressed before embarking on detailed consideration of our results.

Firstly, statistical tests of association assume independent measures which are clearly not the case in our study, since the same source has been used to derive both ICD-8 and DSM-III ratings. Log-linear analysis confirmed that the only significant interaction was between DSM-III and ICD-8 classifications. Thus we felt able to proceed with standard statistical tests of association the χ^2 and kappa statistics. However, the kappa agreement between the two classifications is modest, despite being significantly better than chance according to standard significance measures. Indeed, conventionally, kappa values less than 0.4 are considered poor and Table 2 shows that except for 295/297 and no diagnosis, agreement is clearly below this acceptable limit.

Secondly, the CATEGO4 ratings are not derived from the PSE but from a differently worded subset of CIDI items. While the PSE is a semi-structured interview devised for use by trained clinicians, CIDI is highly structured and specifically devised for use by lay interviewers. Both interviews however require a period of training in their use. While our previous study encouraged us to consider that CIDI-generated PSE ratings approximated well to PSE-generated ratings, they are clearly not the same and our results must be interpreted with this proviso.

In turn, the interpretation of ICD-8 diagnoses via the CATEGO4 program is tentative as ICD-8 contains no explicit rules to guide the construction of diagnostic algorithms. However, the CATEGO interpretation of ICD-8 has received widespread research use internationally and therefore general acceptance. Rather than just compare the end-point of the CATEGO process, namely the tentative ICD-8 assignment, we examined the PSE syndrome frequency for some of the categories of affective disorder. As mentioned in the introduction, 300.4 denotes rather different disorders in ICD-8 and DSM-III. In ICD-8 300.4 is a broad category covering an admixture of depressive and neurotic symptoms often developing acutely in response to a psychosocial stressor, while in DSM-III 300.4 is a dysthymic disorder without anxiety of at least two years' duration. Reference to the cross-tabulation in Table 2 shows that ICD-8 300.4 overlaps with DSM-III 300.0, 296.2, 296.3 and no diagnosis. Fig. 2 shows that the syndrome profile for 300.4 derived from DSM-III and ICD-8 is surprisingly congruent and also similar to Fig. 1 for 296 categories. Reference to the profile data from the International Pilot Study of Schizophrenia (Wing *et al*, 1974) shows that all four profiles resemble the R+ profile rather than that for anxiety (A+). However, Fig. 2 (for 300.4) must be interpreted with some caution since there are only two cases which are DSM-III 300.4.

The results show that the number of cases classed as having schizophrenic psychosis (295) by DSM-III and CATEGO4 remains fairly constant over time. This is probably related to patient selection, as all psychotic subjects included in the study had well established illness. The excess of cases assigned to 295 by CATEGO4 compared with DSM-III probably reflects differences in the coding algorithms for each classification. DSM-III may be considered to have somewhat stricter criteria for the diagnosis of schizophrenia. For instance CATEGO4 does not take symptom duration into account but is highly likely to assign a schizophrenia category solely on the presence of Schneiderian first-rank symptoms (Schneider, 1959), whereas DSM-III requires six months of illness or dysfunction before schizophrenia can be diagnosed. Thus only cross-sectional information is required for a CATEGO4 295 category whereas longitudinal data are necessary for DSM-III schizophrenia.

We have examined the sources of disagreement between the two classifications in order to see if any clear trends emerge. As discussed above, it might be predicted that differences between classifications for type of psychotic disorder might produce the greatest number of discordant cases. Indeed, as Table 2 shows, this source of disagreement produced 21% of discordant cases for current illness rising to 35% for lifetime. Previous reliability studies (Robins *et al*, 1982) have shown that the area of greatest disagreement for inter-rater agreement lies on the border between mild illness and health. Much has been written and debated about the cut-off point that divides 'cases' from 'non-cases' (Wing *et al*, 1978). Thus it might be expected that the greatest area of discord between classifications might lie at the border between defined illness and relative health. While this source accounted for 32% disagreement for current illness, this fell to 8% for lifetime illness. Put more positively, the two classifications agree 97% of the time about what is a definite 'case'.

The greatest number of cases differed according to whether an anxiety state or a 29 diagnostic group was assigned (46% for present state and 58% for lifetime). The kappa analysis showed that bias was directed towards CATEGO4 assigned a 300 class, although this was not statistically significant (for present state bias=27.0, $\chi^2=0.148$, P=NS; for lifetime, bias=26.0, $\chi^2=0.038$, P=NS). Thus CATEGO4 tends to assign what might be considered a 'more severe or 29' diagnostic group compared with DSM-III, which tends towards the less severe or 300 grouping. This corresponds with the former distinction between 'neurotic' and 'psychotic' diagnostic categories which still remain in ICD-8 but which are omitted from DSM-III. The most likely explanation for this finding, as we have already pointed out, is that the coding algorithms for major psychiatric disorders in DSM-III are more stringent than for CATEGO4-derived ICD-8.

Approximately a third of cases are diagnosed as having an anxiety state by both classifications for present illness, and this proportion is maintained over lifetime illness by CATEGO4. However, in DSM-III the number of cases assigned to this group is reduced when lifetime diagnosis is examined, with cases apparently shifting to an affective disorder category. Indeed, for both time frames DSM-III assigns most cases to 296, but particularly for lifetime. In CATEGO4, however, there is a more even distribution of cases across 295, 296 and 300 diagnostic groups. Both these findings suggest that, compared with the CATEGO4 classification, DSM-III produces a diagnostic shift away from the schizophrenic psychoses (295) and anxiety states (300) towards affective disorder and that this is most marked for a lifetime diagnostic time frame.

These findings are at variance with an earlier study (Brink *et al*, 1989) comparing PSE/CATEGO and DSM-III in an out-patient group. These authors also found comparatively low agreement between classifications but showed a bias for PSE/CATEGO diagnosing depression compared with DSM-III. However, a revised version of the PSE was used to make both CATEGO and DSM-III diagnoses, the inter-rater reliability for which is not reported in their study. In addition, these authors also suggest that in their out-patient group, PSE/CATEGO is relatively insensitive in diagnosing social phobias and obsessive-compulsive disorder. Our findings, the reverse of this, suggest that in a more severely ill sample, the DSM-III criteria for affective disorder are broader (Cooper *et al*, 1972; World Health Organization, 1973) than elsewhere. Such differences may have major implications in relation to patient management and statistics, and also for research in mental illness.

Both classifications appear to be somewhat sensitive to the type of subject group under scrutiny. In an out-patient setting, PSE/CATEGO categorises more individuals as suffering from affective disorder compared with DSM-III, while the reverse appears to be true for more severely ill in-patients. In addition, Spitzer & Williams (1988) have pointed out that the interpretation of seemingly quite explicit diagnostic criteria by individual clinicians can be quite different. While some consider schizophrenia as the top of a hierarchy of functional psychoses, other clinicians will place greater emphasis upon affective symptoms in making a diagnosis. Thus far, attempts to examine the relationship between different classifications have been somewhat limited and what research has been carried out indicates that different sets of criteria select cases according to different validating parameters (McGuffin *et al*, 1984; Farmer *et al*, 1987 *b*, *c*).

Although we can draw only limited conclusions from our study, we have attempted to highlight some of the nosological issues which can arise when classifications are compared. CIDI and the CATEGO4 program facilitate the reliable application of different classifications, which is an important gain. However, the absence of validating criteria means that all classifications remain working hypotheses and no single diagnostic schema can be regarded as correct.

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Randomised Controlled Trial of Day and In-patient Psychiatric Treatment. 2: Comparison of Two Hospitals

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Acutely ill patients presenting for admission in two district psychiatric services were randomly allocated to day-hospital or in-patient care. In both services a quarter of all admissions could not be allocated because they were too ill (half of these were compulsory admissions); these patients were predominantly manic and schizophrenic patients with pronounced psychotic symptoms and disturbed behaviour. In one service 80% (41/51) of patients randomly allocated to day-hospital treatment were successfully engaged in treatment compared with 54% (19/35) in the other service. This difference arose because only patients with mildly disturbed behaviour could be treated at the second day hospital. For patients who were successfully allocated, the outcome of treatment was similar in terms of psychiatric symptoms and social functioning. The results of a treatment trial for acutely ill patients in one district service cannot be generalised to other district services without due attention to the factors, such as staffing levels, which determine the degree of disturbed behaviour that can be tolerated in the day hospital.

There has been recent enthusiasm for treating acutely ill patients in a day hospital rather than in an in-patient unit (Department of Health and Social Security, 1975; Vaughan, 1985). However, it has not been matched by a clear demonstration that day treatment is both feasible and efficacious. The early American study of Zwerling & Wilder (1964) showed that two-thirds of all acute admissions could be treated in the day hospital and that such treatment was at least as effective as in-patient treatment. Subsequent attempts to replicate this study failed because representative samples could not be obtained. For example, in the study of Hertz *et al* (1971) only 20% of admissions could be randomly allocated between the two treatment settings, the remainder being excluded. In addition to the problem of unrepresentative samples, there have been other methodological problems that have led to marked criticism of all previous studies (Wilkinson, 1984; Creed *et al*, 1989*a*).

In the UK there have been two attempts to compare day and in-patient treatment, but neither successfully allocated an adequate number of acutely ill patients. One was abandoned because clinicians would allow only 10% of all admissions to be allocated (Platt *et al*, 1980): the majority of patients were regarded as 'mandatory' in-patients (often because they were psychotic or suicidal) or 'mandatory' day patients because they were not ill enough to merit in-patient admission. The other (Dick *et al*, 1985) studied only patients with diagnoses of neurotic illness, adjustment reaction and personality disorder; these amounted to only 22% of all admissions.

On the basis of our previous finding that many acutely ill patients could be satisfactorily treated in the Manchester Royal Infirmary Psychiatric Day Hospital (Creed *et al*, 1989*b*), it was possible to perform a randomised controlled trial comparing admission to this day hospital with the Infirmary's in-patient unit (Creed *et al*, 1990). An identical trial was run simultaneously at a nearby hospital with a different model of day-hospital treatment. This was at Blackburn, whose psychiatric service, like many others, had tended to use the in-patient unit for acutely ill patients and the day hospital either for the treatment of less ill patients or as a rehabilitation unit (Pryce, 1982; McGrath & Tantam, 1987).

There are other differences between the two hospitals and their catchment populations. Although both day hospitals are similar in size (50 day places), Manchester had eight nurses and three occupational therapists at the time of the study whereas Blackburn had six nurses and no dedicated occupational therapists (patients attended the occupational therapy department as necessary). Both day hospitals use individual treatment plans for their patients and offer a full range of treatments. Central Manchester Health District is a socially deprived inner-city area in which all patients live within 3 miles (2 km) of the day hospital. Blackburn is a semirural district with some people living more than 20 miles (32 km) away from the day hospital.

The additional trial at Blackburn allowed us to compare results between the two centres and to infer whether the Manchester findings could be generalised to other district psychiatric services. The specific aims of the present study were to answer the following questions regarding each hospital service.

(a) What proportion of acutely ill patients could be randomly allocated to either day or in-patient treatment?
(b) What were the characteristics of those patients for whom day-hospital treatment was not considered feasible?
(c) What was the clinical and social outcome for patients treated in the day hospital compared with that of those in the in-patient unit?

This study involved two departures from routine clinical practice. Firstly, all patients who required psychiatric admission were considered for the random-allocation procedure; patients admitted as out-of-hours emergencies to the in-patient unit and subsequently randomly allocated to day-patient treatment were rapidly transferred to the day hospital. Secondly, once a patient had been allocated to a treatment setting, the responsible consultants were requested, wherever possible, to maintain the patient in that setting until discharge to out-patient status. At Manchester three out of six consultants who admit to general psychiatry beds were involved; at Blackburn two of four were involved.

The design of the study has been described in detail by Creed *et al* (1990); it was necessary to accept from the outset that some patients would not be available for random allocation (patients admitted under a section of the Mental Health Act, otherwise too ill, or for social reasons). For the patients who were randomly allocated, comparison of outcome of treatment is possible only if the baseline data indicate that the day-patient and in-patient groups were similar in all respects. This was only achieved at Manchester, so those results have been published separately (Creed *et al*, 1990) as an intention-to-treat comparison (*Lancet*, 1987). Since there were clear differences between the patients randomly allocated to day and in-patient groups at Blackburn this paper is *not* intended as a treatment-outcome study but as a description of the patients who were eventually included in the study at the two hospitals. Details of the factors responsible for the differences, notably attitudes of those involved, are explored in a different publication (Anthony *et al*, 1991).

Method

All patients aged 18–65 years who required admission a either in-patients or day patients under the care of one c the five consultants were eligible for inclusion. Both urgen and planned admissions were included, the only exclusion being those patients admitted solely for detoxification fror drugs and those who discharged themselves before the could be considered for random allocation. Three group of patients were studied: (a) those randomly allocated t day treatment, (b) those randomly allocated to in-patien treatment, and (c) all patients not excluded who we admitted during the study period but who could not tak part in the random-allocation procedure because they wer too ill to be treated as a day patient or because the were admitted under a section of the Mental Healt Act.

Before each admission, the consultant in charge decide whether the patient was suitable for random allocation. Fo patients admitted directly to the in-patient unit a emergencies outside normal working hours, the decisio was taken on the next working day.

Allocation was by taking a card from a sealed envelop which indicated day or in-patient treatment.

On admission and at three months and one year afte admission, mental state was assessed using the Present Sta Examination (PSE; Wing *et al*, 1974). This provide syndromal scores for the symptoms present during th month before admission and a tentative ICD diagnosis

Social functioning was assessed at the same points in tim using the Social Behaviour Assessment Scale (SBAS), whic yields scores for social role performance (e.g. househol management, employment, spare-time activities), an abnormal behaviour (e.g. withdrawal, odd ideas, ove activity) and burden on relatives (e.g. lost sleep, tim off work or disrupted household routine) (Platt *et al*, 1981 On admission, these measures were made for two period of time: immediately before admission ('when ill'); and fo the time before the recent illness when the person wa considered to be his/her normal self ('when well'). Th assessment required detailed information from a clos relative or other household member (or hostel warden). could therefore be made only when such an informant wa available.

On admission, demographic data were collected fror every patient. Five days after admission, the patient' behaviour in the hospital was assessed using a modifie form of the Social Behaviour Scale (SBS; Wykes & Stur 1986). This is a standardised measure, with clearly define points, of the patient's behaviour observed by th responsible nursing staff.

The PSE results were analysed using the CATEG program. They are presented here as the four sum mary scores: delusions and hallucinations (DAH behaviour, speech and other syndromes (BSO), specifi neurotic reaction (SNR), and non-specific neuroti symptoms (NSN). The SBAS is expressed as three summar scores: role performance, abnormal behaviour, and burde on relatives. The SBS results are presented both as score for individual behavioural ratings and as a summar score.

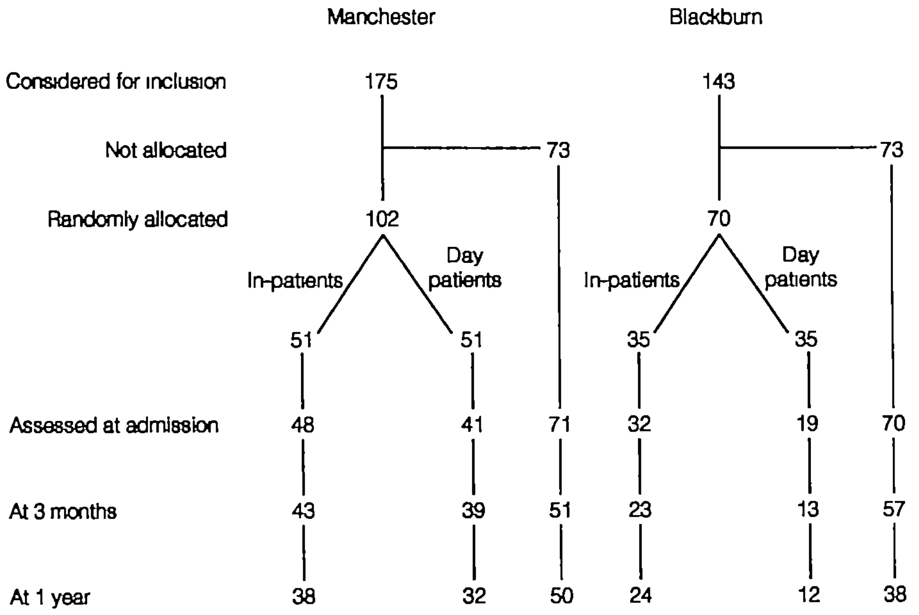

Fig. 1 Number of patients in each group and number assessed at each time point.

Statistical differences between groups were assessed using ıe Mann–Whitney, χ^2 (with Yates' correction) and ruskal–Wallis one-way ANOVA tests.

Results

he numbers of patients considered for inclusion in the udy were 175 at Manchester and 143 at Blackburn. Of ıese, 102 (58%) were randomly allocated at Manchester nd 70 (49%) at Blackburn ($\chi^2=2.39$, NS). However, not ll patients remained in their allocated treatment modality ɔr the full initial assessment: three in-patients at lanchester and three at Blackburn discharged themselves efore the research interview could be performed, and 10 ay patients at Manchester and 16 at Blackburn failed to ttend consistently (Fig. 1). Compared with Blackburn, ignficantly more of the Manchester patients became stablished in their allocated form of treatment (89/102 v. 1/70, $\chi^2=4.77$, $P<0.05$). This difference ceases to be ignificant if allowance is made for the six Manchester atients who had to be later transferred from day hospital ɔ the in-patient unit (Creed *et al*, 1990).

Of all allocated and non-allocated patients, complete ıental-state assessments were obtained at admission on 160 f the 175 Manchester patients and 121 of the 143 llackburn patients. At three months and one year, further 'SEs were recorded for 133 (83%) and 120 (75%) of the lanchester patients, and for 93 (77%) and 74 (62%) of the Blackburn patients. Of the drop-outs from both centres, 49 patients were untraceable (many were known to have moved away), 35 refused further interviews, and three had died or developed severe dementia.

Complete SBAS interviews were obtained from informants of 132 patients at Manchester and 98 at Blackburn. The numbers reinterviewed at three months and one year were 112 (85%) and 103 (78%) at Manchester, and 85 (87%) and 77 (79%) at Blackburn.

Differences between the Manchester and Blackburn samples

As shown in Table 1, the Manchester and Blackburn samples were not identical on demographic measures. The Manchester sample had significantly more men, more non-Caucasians, more patients who lived alone, and fewer patients who were admitted as an emergency. The Blackburn patients had experienced more previous psychiatric admissions and had a longer present episode of illness. There were no significant differences in age, country of birth, or age of onset. In terms of diagnosis the samples were similar (Table 2).

Patients not randomly allocated

As shown in Table 3, the two centres did not differ significantly in the proportions of patients who could not be randomly allocated for various reasons. Moreover, the

Table 1
Comparison of Manchester and Blackburn patients: demographic data

	Manchester (n=160)	Blackburn (n=121)
Mean age. years	40.6	41.0
% female	43%	57%*
% single	47%	37%
% non-Caucasian	18%	4%**
% off sick	36%	26%
% living alone	37%	19%*
% currently ill >1 year	17%	32%**
Mean no. of previous admissions	2.1	2.9*
% referred from accident and emergency	12%	28%**
% emergency admission	68%	79%*
% on section	11%	12%

*$P<0.05$, **$P<0.01$ (χ^2 for % values, Mann–Whitney U test for age and no. of admissions).

clinical and social characteristics of the patients not allocated at each centre were similar. For the purposes of comparing non-allocated and allocated patients the Manchester and Blackburn samples have therefore been combined.

There was a significant difference in diagnostic categories between non-allocated and allocated patients. A total of 50% of the patients not allocated were schizophrenic or manic, compared with 37% of the randomly allocated patients (Table 4). However, the PSE syndrome profiles, SBAS summary scores and SBS scores were not significantly different when all the non-allocated patients were compared with the randomly allocated group.

The non-allocated patients were then divided into three subgroups according to reason for non-allocation (Table 5). The patients admitted compulsorily had high scores for both psychotic symptoms (DAH) and behavioural disturbance (BSO and nurses' rating). The patients considered 'too ill' had high scores only for psychotic symptoms, and those who were not allocated for other reasons (social reasons and refusal) had symptom and behavioural scores similar to the randomly allocated group.

Of the three sets of measures, the SBS demonstrated the clearest differences between allocated and non-allocated patients. Fig. 2 demonstrates the results for the nurses' ratings of those individual items of behaviour that showed greatest difference between allocated and non-allocated patients. It is also apparent from Fig. 2 that at Blackburn, but not at Manchester, the randomly allocated day-hospital patients were less disturbed in their behaviour than the randomly allocated in-patients. This finding is also apparent in the summary scores for all 24 nurses' ratings (a high score indicates more disturbed behaviour). At Manchester and Blackburn the summary scores for non-allocated patients were similar: median and range = 12 (0–41) and 11 (0–41) respectively. Those for randomly allocated in-patients were also similar: 6 (0–25) and 8 (0–27) respectively. But for randomised day patients there was a significant difference: 7 (0–22) for Manchester and 2.5 (0–12) at Blackburn ($P<0.05$). It can be seen that the randomised day and in-patients had similar median scores and ranges at Manchester but at Blackburn the day patients had a lower median score than the randomised in-patients and upper range of approximately half the value.

The scores of the six patients who were randomly allocated to the day hospital but who were transferred to in-patient care had a median SBS score of 14 (range 2–20) at Manchester, indicating that they were as disturbed as the non-allocated patients. At Blackburn there were no such transfers because these patients had not become established in day-hospital treatment at the outset. But the median score for the patients randomly allocated to day-hospital treatment who did not become established in that form of treatment was 8.5 (range 0–24), which was similar to that of the randomised in-patients. Had they been successfully engaged in day treatment that group would have been similar to the Manchester day-hospital group.

Table 2
Diagnostic categories by centre and group

	Manchester					Blackburn				
	randomly allocated		non-allocated	total	%	randomly allocated		non-allocated	total	%
	in-patients	day patients	(in-patients)			in-patients	day patients	(in-patients)		
Schizophrenia	14	10	26	50	31%	10	2	20	32	26%
Depression	10	8	15	33	21%	6	4	17	27	22%
Mania	4	4	13	21	13%	4	3	12	19	16%
Neurotic disorders	13	11	2	26	16%	2	5	6	13	11%
Personality disorders	5	3	8	16	10%	5	1	9	15	12%
Addiction/ organic disorders	2	5	7	14	9%	5	4	6	15	12%
Totals	48	41	71	160		32	19	70	121	

Table 3
Reasons for non-allocation

	Manchester ($n=71$)	Blackburn ($n=70$)
Under section of MHA	17 (24%)	16 (23%)
Considered too ill	28 (39%)	17 (24%)
Refusal	4 (6%)	7 (10%)
Social reasons	8 (11%)	15 (21%)
Other	14 (20%)	15 (22%)

$\chi^2=5.69$, d f. $=4$, NS.

Table 4
Number of patients in each diagnostic category according to randomly allocated and non-allocated groups (Manchester and Blackburn samples combined)

	Randomly allocated	Not-allocated
Schizophrenia	36 (26%)	46 (33%)
Depression	28 (20%)	32 (23%)
Mania	15 (11%)	25 (17%)
Neurotic disorders	31 (22%)	8 (6%)
Personality disorders	14 (10%)	17 (12%)
Addiction/organic disorders	16 (11%)	13 (9%)
Totals	140	141

$\chi^2=18.1$, d.f. $=5$, $P<0.005$.

Table 5
Comparison of patients not allocated because of admission under Mental Health Act, voluntary patients considered too ill for random allocation and those not allocated for other reasons, with scores for patients who were randomly allocated given for comparison

	Patients not allocated			Patients randomly allocated ($n=140$)
	admitted under MHA ($n=33$)	considered too ill ($n=45$)	other reason ($n=63$)	
PSE				
DAH	3.4^+	3.0	1.6	1.8^*
BSO	2.7^+	1.5	1.1	1.1^*
SNR	1.6^+	5.5	2.7	4.1^*
NSN	4.8^+	10.0	7.5	9.8^*
SBAS				
Role	15.1	14.1	12.7	13.4
Burden	5.6	6.0	5.0	5.2
Behaviour	16.4	16.0	15.0	15.3
SBS				
Nurses' rating	14.9^+	13.7^+	9.9	8.8^*

$^*P<0.001$ (Kruskal–Wallis) across all four groups.
$^+$Indicates significant difference ($P<0.01$) from randomly allocated group
DAH = delusions and hallucinations, BSO = behaviour, speech and other syndromes, SNR = specific neurotic reactions, NSN = non-specific syndromes.

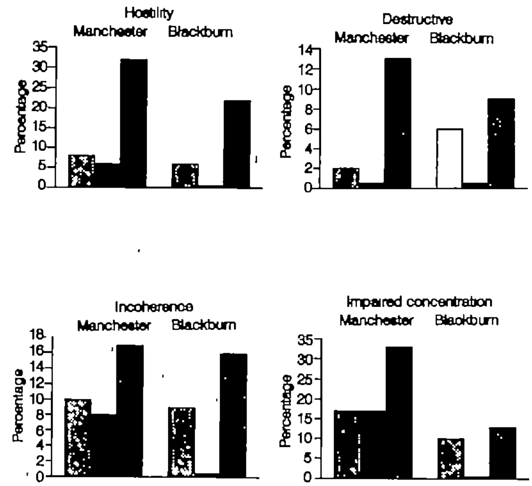

Fig. 2 Nurses' rating of behaviour on the SBS subscale: Percentage in each group of patients given a rating of two or more. Items shown are those which showed greatest difference between non-allocated and all randomly allocated patients. Data are shown for randomly allocated in-patients ▨, randomly allocated day patients ■, and non-allocated patients ▩, for Manchester and Blackburn separately.

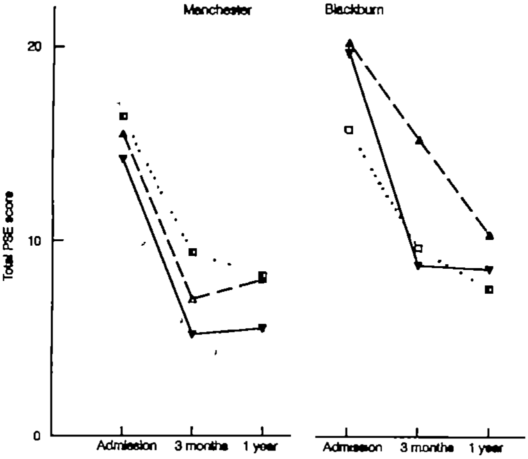

Fig. 3 Reduction of total PSE score between admission, three months and one year (□····□ day patients, △—△ non-allocated patients, ▽—▽ in-patients).

Effect of treatment

The results of the Manchester treatment study have been published elsewhere (Creed *et al*, 1990). The results of the Blackburn study cannot simply be added to these because the random-allocation procedure did not produce equivalent groups of patients at each centre.

The overall reduction of psychiatric symptoms at three months and one year was similar in all groups, suggesting that the effect of day-hospital treatment was equivalent to that of in-patient care (Fig. 3). Detailed examination of the results was made using an 'improvement score' calculated by subtracting the scores at three months from the scores at admission. There were no significant differences between day and in-patient treatment groups for DAH, BSO, SNR, and NSN scores of the PSE or behaviour and burden subscores on the SBAS. The only significant difference was in role performance at three months in the Manchester sample – in-patients had improved to a greater extent ($P<0.05$).

There were no significant differences between day and in-patients at one year. Since one-fifth of patients were lost to follow-up at three months (one-quarter at a year), the patients not followed up were compared with the whole cohort. They had not been significantly different on any PSE, SBAS or SBS measures at initial assessment. The results for the 116 patients who were interviewed on all three occasions showed no significant difference between day and in-patients at one year. The details of readmission rates will be published separately.

Discussion

The aim of this project was to run a random-allocation study, comparing the effectiveness of day-hospital and in-patient treatment, at two centres. The design allowed for the following inevitable constraints. Firstly, patients admitted under a section of the Mental Health Act or others who were seriously disturbed could not be considered for day treatment and therefore would not be available for random allocation. Secondly, the day hospitals at Manchester and Blackburn might not be able to admit similar cohorts of patients because of different staffing levels and prior uses of day-hospital facilities (Creed *et al*, 1989*b*). Because the groups of patients successfully allocated to day treatment differed at the two centres these must be regarded as two parallel studies rather than a single one. For reasons given above, the Blackburn study cannot be regarded as an intention-to-treat comparison.

In the event, both centres were able to randomly allocate a larger proportion of patients (58% and 49%) than some previous studies (10–22%) (Hertz *et al*, 1971; Fenton *et al*, 1979; Platt *et al*, 1980; Dick *et al*, 1985). However, only 19 patients of the 35 patients randomly allocated to the day hospital at Blackburn were successfully engaged in that form of treatment. If we assume that a corresponding 19 patients of the randomised in-patient group could also have been treated in the day hospital, 38 patients (31%) of the 121 patients assessed in this project might be treated in that day hospital. The corresponding figure for Manchester was 51%.

The feasibility of day care for acutely ill patients (Creed *et al*, 1990) has therefore been partially demonstrated at a second day hospital. Unlike Dick *et al* (1985) and Platt *et al* (1980), we found it possible to treat a number of psychotic, disorganised and suicidal patients in the day hospital; the extent of this depended on the particular day hospital.

The characteristics of those patients who cannot be treated in a day hospital have been defined for the first time in the UK. They are not, however, a homogeneous group and two groups can be discerned: (a) patients who are too ill (including those admitted under the Mental Health Act) and (b) patients for whom random allocation is not possible because of social reasons, for example lack of suitable accommodation, or refusal by patients or others involved in the patients' care (either family or social workers).

There were almost twice as many patients in category (b) at Blackburn compared with Manchester, even though more Manchester patients lived alone. This suggests that attitudes of patients, relatives and staff may have been important in determining suitability for random allocation. At Manchester the day hospital is a smart new building on the site of a district general hospital and most of the in-patient beds are seven miles (11 km) away in a psychiatric hospital. At Blackburn the two units are on the same site and the prevailing attitude, particularly among patients and their relatives, is that the in-patient unit is where 'proper' psychiatric treatment takes place.

At each hospital a research registrar was employed to screen all admissions and discuss them with the relevant consultant. This prevented the junior staff (whom Platt *et al* (1981) found to be more reluctant than senior staff to allocate patients randomly) influencing the proportion so allocated.

Staff numbers may be more important than attitudes. The greater behavioural disturbance that could be managed in the Manchester day hospital probably reflects greater staff numbers, as more time can be given to disturbed patients. This would reduce the chances of disturbed behaviour leading to demands for transfer to in-patient care. These points will be considered in more detail in another publication.

The outcome of day and in-patient treatment was similar at the two centres. The main qualification to this statement is the small number of patients successfully completing a course of day-hospital treatment at Blackburn. The number of subjects reassessed at three months and one year was disappointing, but our follow-up rate is comparable with that in our previous study (Creed *et al*, 1989*b*) and rather better than some similar studies (Creed *et al*, 1989*a*). The figures reflect the reality of accepting into the study *all* admissions, not only those living with a stable family (Hertz *et al*, 1975). Whether there were lasting differences between day and in-patient treatment in readmission rates over a longer follow-up period will be the subject of another paper.

This study has demonstrated that day-hospital treatment is an alternative to in-patient treatment for acutely ill patients but adequate numbers of staff and confidence among the staff, patients and their relatives are necessary if the proportion is to reach 50%. Any increase in this proportion would require 24-hour community nursing to help relatives and other carers to deal with a patient's disturbed behaviour out of hours (Stein & Test, 1980; Hoult, 1986).

The number of staff required for satisfactory day-hospital treatment of acutely ill patients means that day care may not be cheaper than in-patient care (Weisbrod *et al*, 1980). It may be a preferable form of treatment if accepted by patients and their relatives and if it reduces subsequent readmission rates (Creed *et al*, 1990). On the other hand, there may be increased burden on relatives during the early stages of treatment which was not examined in detail during the present study. There may also be additional costs to general practitioners and other carers in the community, which at present remain 'hidden'. All these aspects of day-hospital treatment are being examined in our current study.

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Planning for Community Care

Long-Stay Populations of Hospitals Scheduled for Rundown or Closure

P. CLIFFORD, A. CHARMAN, Y. WEBB and S. BEST

Using the Community Placement Questionnaire, the long-stay populations of five hospitals were surveyed. The results suggest that there is little need for large hospitals if adequate community provision is made. However, a small number of patients continue to accumulate for whom community placement is hard to envisage. Investigating the characteristics of the 'new long-stay' patients suggests that the usual definition should be extended to include those over 65 years old with no diagnosis of dementia and those in hospital for 1–10 years. About 20% of 'new long-stay' patients have organic diagnoses and the needs of this group require assessment.

The majority of districts with patients in long-stay psychiatric hospitals are currently involved in developing community-based facilities for a significant proportion of their long-stay population. In many cases hospitals have closure dates; in most others the intention is to reduce the number of available hospital long-stay beds considerably over the next decade. As the idea of hospital closure moves closer to becoming a reality, debate has intensified over what range of facilities and support is required for the current population once discharged into the community. Worries about the lack of provision being made and also about the appropriateness of community placement for some of the most disabled patients have led to calls either for a halt to the hospital closure programmes (National Schizophrenia Fellowship, 1989) or for the development of smaller asylums or 'havens' (Wing & Furlong, 1986) on hospital sites. There is also a growing awareness of the problems posed by the so-called 'new long-stay' population (Levene *et al*, 1985), usually defined as those people under 65 years old who have been in hospital for between one and five years (Mann & Cree, 1976). Many of these patients require high levels of support for prolonged periods and this has led to the development of a new type of specialist facility, the 'hostel ward' (Garety & Morris, 1984).

In this context many districts have undertaken surveys of their hospital populations with a view to establishing the patients' needs were they to be discharged. There is agreement that the residual hospital population is characterised by high levels of disability and a corresponding need for high levels of staff support (Christie-Brown, 1977; Ford *et al*, 1987). However, since Mann & Cree's (1976) study of the new long-stay populations in 15 hospitals there has been a shortage of studies comparing hospital populations. This makes it hard to generalise from published findings and brings with it the danger that the finer detail of population trends that can emerge from larger studies may remain unidentified because of the relatively small sample sizes involved.

This article presents findings from surveys of five psychiatric hospitals involved in planning for community care. The surveys were carried out over two and a half years, beginning in November 1985. The results are used to consider the characteristics of the new long-stay population and the need for asylum.

Method

The surveys covered all patients without a diagnosis of senile dementia who had been in hospital for more than one year. The final sample comprised 1308 patients (635 men, 673 women). The hospitals all had between 275 and 330 long-stay patients resident for more than one year.

Information was collected using the Community Placement Questionnaire (CPQ; Clifford, 1989), a standardised staff-completion schedule designed for service planning. The schedule includes demographic data, and ratings of social functioning, problem behaviour, physical disability, social contact and needs for accommodation and day care in the event of placement in the community. Reliability and validity are described elsewhere (Clifford *et al*, 1990). Overall, the schedule fulfils Hall's five minimum criteria for ward rating scales for long-stay patients (Hall, 1980). (Full details of ratings, scoring procedure and of the CPQ's standardisation are available from the authors.)

Results

The population was predominantly elderly with an average age of 64.5 years. Two-thirds were aged over 60 and two-fifths were over 70. Only 3% were aged under 30, 9% under

Table 1
Patient characteristics and placement recommendations

Placement recommendations[1]	Mean age: years	Mean length of stay: years	Social functioning[2]	Physical disability[2]	Behavioural problems	Psychiatric impairment[2]
Independent (*n* = 39)	51.5	6.7	2.9	1.4	1.0	1.2
Low dependency (*n* = 330)	60.5	19.7	2.8	1.5	1.4	1.5
High dependency (*n* = 885)	66.5	25.5	2.4	2.1	2.2	2.7

1. 54 patients recommended to 'other' placements are not included in Table.
2 Scores from Community Placement Questionnaire items.

40, and 18% under 50. The average length of stay was 24.5 years. Half the population had been in hospital for over 20 years, a fifth for over 40 years and a fifth for under five years. Age and stay did not necessarily go hand in hand: almost a quarter of those aged over 65 had been in hospital for less than ten years; and only 9% of the population were under 40 years old but a third of these had been in hospital for less than ten years.

Seventy per cent of both men and women had a primary diagnosis of schizophrenia. Twelve per cent had a diagnosis of either affective psychosis or neurotic depression, two-thirds of these being female. Diagnosis of schizophrenia was closely associated with greater length of stay and diagnoses of affective psychosis with shorter length of stay: 56% of patients admitted in the past five years had a diagnosis of schizophrenia and 20% had an affective disorder, compared with 72% and 7% respectively of those with a stay of 20–30 years and 91% and 2% of those with a stay of over 40 years.

Forty-one patients (3%) had a primary diagnosis of mental retardation and 85 (7%) had other organic syndromes such as epilepsy or 'other organic psychosis' – a category that includes Korsakoff's syndrome, brain damage and Huntington's chorea. One-fifth of the population had a primary or secondary 'organic' diagnosis, three-quarters of these being male. Organic diagnoses were twice as frequent among patients with a stay of one to five years than among those with a stay of over 20 years.

More than half of the patients needed supervision with basic activities of daily living, such as dressing and personal hygiene, and the vast majority were unable to shop or cook for themselves or use public transport unaided. However, two-fifths had a 'moderate' or 'high' level of functioning. Over half were rated as having psychiatric disabilities that significantly limited their participation in rehabilitation programmes or social activities.

Two-fifths had behavioural problems thought likely to present significant difficulty in a community setting, half of these of a severity considered likely to be hard to manage in a community setting. About 10% of patients were rated as being a significant or serious danger to themselves or others. About 10% had a recorded history of violent or dangerous behaviour, although many such incidents occurred in the distant past and may be of questionable relevance.

Thirty-two per cent had a significant level of physical disability, of which about half were rated as having a serious effect on functioning. Of the total population, 25% had problems with mobility and 30% were incontinent weekly or more.

Levels of disability did not vary significantly across hospitals or in relation to sex or length of stay.

Needs for care

The accommodation item on the CPQ comprises eight options graded according to level of supervision required. These can be collapsed down into bands of 'independent', 'low-staffed' and 'high-staffed' homes. The recommendations are shown in Table 1. Only 39 patients were recommended for independent placement, half of these to return to their families and the remainder to independent flats or group homes. Twenty-seven per cent were recommended for low-staffed placement, about two-thirds of these to low-staffed homes and most of the remainder to supported group homes. Seventy-one per cent were recommended for high-staffed placement. About one-third of these were recommended for very sheltered nursing homes and the remainder to high-staffed homes with 24-hour staffing comparable with hospital levels. Fifty-four patients were recommended for 'other' placements including secure units and specialist units for people with brain injury.

Disability on all measures increased as patients were recommended for more dependent forms of accommodation. Behavioural problems and psychiatric disability were the most important determinants of staff recommendation, with differences in social functioning also distinguishing those recommended for 24-hour placement from those judged to need lower staffing levels. Age and stay were only mildly related to placement recommendation.

On a separate item on the CPQ staff judged that hospital placement would be the most appropriate long-term placement for about a quarter of the population even if community facilities were available. For those admitted in the most recent period of ten years, a rate of about 1.5 per hospital per year were recommended for permanent hospital placement. One-tenth of those with a stay of less than five years were recommended for hospital placement, compared with 22% of those with a stay of 10–20 years and 39% of those with a stay over 40 years. Only 13% of those under 50 years old were recommended for long-term hospital placement.

Overall, the people recommended for hospital placement were typically older (two-thirds were aged over 65), more

Table 2
Patient characteristics by recommendation for hospital placement

	Recommendation	
	Hospital placement ($n = 320$)	Community placement ($n = 988$)
Mean age: years	69.8	62.8
Mean length of stay: years	31.3	21.1
Social functioning[1]	2.2	2.6
Physical disability[1]	2.5	1.8
Social unacceptability[1]	2.9	1.6
Psychological impairments[1]	3.4	2.1
Dangerousness[1]	1.1	0.5

1. Scores from Community Placement Questionnaire items

disabled on all measures, and usually multiply handicapped (Table 2). They constitute one set of candidates for 'asylum' care.

Twenty-eight per cent of the population studied were assessed as 'hard to place' using a composite measure that is independent of staff ratings of needs for community placement (Clifford *et al*, 1990). Their average age was 59.7 years and average length of stay 21.3 years. A third of those with a stay of less than ten years were classified as 'hard to place' as were nearly half those under 50 years old.

Ninety per cent of these patients had serious behavioural problems, half had poor social functioning, a quarter had serious physical disabilities and a third had had to be contained in a secure environment in the preceding two years. Over three quarters were recommended for placements with 24-hour staffing.

Those with organic diagnoses contained a higher proportion of people who were hard to place (37% as against 26%). Compared with patients with a diagnosis of schizophrenia or affective psychosis these patients tended to be less able to participate in structured activities or rehabilitation programmes and to have higher levels of physical disability and behavioural problems. There was a related tendency to recommendation for placement with 24-hour staffing (over 80% compared with 66%); and a third were recommended for long-term hospital placement.

The 'hard to place' measure is a 'softer' indicator of the 'need for asylum' than the hospital placement recommendation. Indeed, it covers a rather different population – only about 50% of the patients recommended for hospital placement came out as 'hard to place'. This is because the measure puts a lower weighting on age, length of stay and physical disability than that suggested by staff ratings of need for hospital placement.

Ninety-six per cent of the population were considered likely to need some form of day care if moved into the community, over three-quarters of these being recommended for 'low-key' non-pressurising environments and the remainder for day settings involving more intense rehabilitation or socialisation. Only 1% were thought potentially capable of open employment at the time of the survey and a further 11% were recommended for sheltered work settings if they were available.

The 'new long-stay population'

A subpopulation of particular interest are those patients who have been admitted in recent years and who have remained in hospital despite the policy of ensuring discharge to the community wherever possible. The analysis adopts the definition of 'new long-stay' as patients who are aged under 65 and have been in hospital for one to five years, except where otherwise stated.

There were a total of 166 such 'new long-stay' patients. About one-third of these were under 35 years old but the average age of the group was 45 years, rather higher than might have been expected from what is often considered to be a predominantly young population.

The number of new long-stay patients rises to about one in five if those aged over 65 are included. In addition, 23% of the population were under 65 years old and had been in hospital for less than 10 years; this proportion rises to 34% if those aged over 65 years are included. Patients admitted relatively recently and often not classified as 'new long-stay' either because of age or length of stay thus represent a significant portion of the hospitals' population.

If those aged over 65 are included, two distinct subpopulations of new long-stay patients emerge, the first being predominantly composed of young male schizophrenics and the second of elderly women with affective disorders (Table 3). The latter population, in particular, tend to be little remarked upon. These diagnostic differences remain in the 1–10-year group.

Table 3
Subpopulations with a stay of 1–5 years

	Age	
	<35 years ($n = 56$)	>65 years ($n = 73$)
Schizophrenia	49 (88%)	23 (31%)
Affective disorder	2 (4%)	31 (42%)
Organic	2 (4%)	12 (16%)
Other	3 (5%)	7 (10%)
Male	37 (66%)	25 (34%)

The main difficulties of the new long-stay population were weighted towards behavioural problems – nearly 40% were 'hard to place' but only 20% had levels of social functioning likely to require intensive staff supervision; and 30% had been contained in a secure environment in the previous two years. Otherwise, patterns of disability were similar to those of patients with longer stays.

About half the new long-stay population were recommended for placement with 24-hour staffing, a fifth for independent living and the remainder to low-staffed accommodation. On the separate question concerning long-term hospital placement this was thought preferable for 15 people, less than 10%.

Obtaining generalisable rates of accumulation is complicated by big differences between hospitals: of the three hospitals for which accurate catchment area populations could be estimated, two had rates of accumulation of about 4.0 per 100 000 per annum (using the 1–5-year

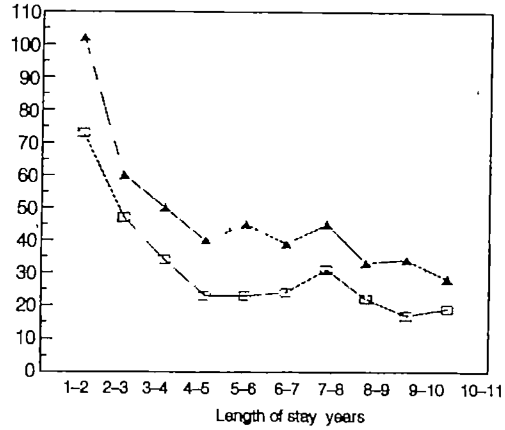

Fig. 1

band in the calculation) and the third a rate of less than half that value. No easy demographic explanation was evident. The accumulation rates in the five hospitals surveyed are shown in Fig. 1.

There is evidently a 'bulge' of people who have been in hospital one to three years, most of whom will be discharged. However, the numbers then flatten off, suggesting that once in hospital for about three years the chances of discharge have become much smaller. This bulge has a big impact on estimates of rates of accumulation: if rates of accumulation are estimated on the basis of those who have been in hospital one to five years the rate is approximately 2.9 per 100 000 per annum; if those staying three to seven years are taken it is 2.1, and if those staying four to eight years are taken it goes down to 1.8.

If the number of patients staying for three years, the point at which the bulge evens out, is taken as indicative of the need for a permanent bed (whether in hospital or elsewhere) this would give a projected need of 8.4 places per 100 000 – a figure strikingly similar to Mann & Cree's (1976) 8–9 places per 100 000 (p. 615).

Rate of accumulation of 'new' patients requiring long-term hospital care

An alternative, and stricter, method of estimating need for permanent beds is to use the clinical team's recommendations for long-term hospital placement as the basis for the calculation. This is likely to underestimate the true numbers of newly accumulating patients who will require long-term hospital care because staff are highly unlikely to recommend someone for long-term hospital placement early on in hospital stay. This underestimate is further compounded by the 'bulge' – the artificially large numbers of patients in the group staying in hospital for one to five years. However, since it is desirable to put a figure to this question, a calculation was made that included all newly accumulated patients with a stay of one to seven years. Extending the range of length of stay from five to seven years compensates partly for the 'bulge'. Using this method there was a rate of accumulation of about 1.9 per 100 000 over the six years, about 0.3 per 100 000 per annum in those under 65 years old. The figure is raised to about 0.4 if those over 65 years old are included.

Discussion

The study is subject to methodological limitations: the hospitals were not selected to be representative of British hospitals, and the surveys were primarily a planning exercise rather than a piece of research. Nevertheless, the size of the sample and the similarities in results across hospitals suggest that they are fairly typical of hospitals planning for rundown or closure. It is therefore worth considering the policy implications of the main findings.

The study confirms the view that psychiatric hospitals in the UK now contain a predominantly elderly and severely disabled population, many of whom have been in hospitals for very long periods. They also confirm Mann & Cree's (1976) observation of striking similarities across hospitals (p. 68). This similarity, coupled with the lack of variation in disability according to sex or stay, suggest that hospitals are consistently discharging their more able patients and only retaining those whose disabilities are of a certain type and severity. This is what would be hoped, but it is reassuring, nevertheless, to see confirmed. However, the data do not support the view that the population is exclusively a 'residual' population. One in three had been in hospital for less than ten years, showing that in the absence of alternative community-based services patients are continuing to accumulate.

The study also echoes the findings of Mann & Cree (1976) and Cumella *et al* (1988) that although by far the highest proportion have a diagnosis of schizophrenia, there are a substantial number of patients with a diagnosis of mental retardation and/or other organic syndromes. There has been little study of this population. Awareness of it tends to be expressed mostly in the form of arguments about which service should take responsibility for these patients (e.g. mental handicap or mental illness, psychogeriatric or mental illness). However, these patients were significantly more disabled and less receptive to rehabilitation programmes than those with other diagnoses. Whether a proportion require separate or specialist facilities therefore requires consideration.

The 'new' long-stay

The data suggest that usage of the term 'new' long-stay needs to be reviewed. The term usually refers to patients with a stay of less than five years and an age of under 65 years (Mann & Cree, 1976). In planning services, this terminology can lead to a tendency to identify the new long-stay with younger patients and the old long-stay with older patients. However, this is misleading: there are a significant proportion of patients who are not elderly but have been in hospital for some considerable time; or who are over 65 years old but have been in hospital for relatively short periods. In addition, there are few differences between the population with a stay of one to five years and those with a stay of five to ten years. Since there are also few differences in levels of disability between the old long-stay and new long-stay populations it could be suggested that the distinction between these be abandoned altogether. However, there is justification on grounds other than disability. The new long-stay population has a higher proportion of patients with affective psychoses and 'organic' diagnoses, and seems to contain at least two distinct subpopulations: the young (mostly) male schizophrenics and the old (mostly) female depressives.

If it is to be used at all, the term 'new long-stay' should therefore be extended to include patients with a stay of less than ten rather than five years, and non-dementing patients over 65 years old. Apart from the high proportion with affective diagnoses, the latter group included schizophrenics who were remaining in hospital because they had become physically frail, or because their elderly relatives were now dead or too frail to look after them. The older populations of new long-stay are inadequately catered for at present, usually getting 'stuck in the joins' between separate planning groups for the elderly and the mentally ill. The result is frequently inappropriate placement. As with the organic group, their needs require proper consideration.

The flattening out of the accumulation curve of new long-stay patients confirms the clinical observation that there is a 'fast' and a 'slow' stream of new long-stay patients, the majority of the former being discharged after three to four years and the latter remaining in hospital for much more extended periods.

The recommendations for 24-hour staffing lend some support to the advocacy of hostel wards for the younger new long-stay population. It is important to include the needs of similarly disabled patients in the community in planning facilities for this group.

Overall, the data on the new long-stay population show it to be more diverse than is often recognised, implying that a wide range of facilities is likely to be required to cater for diverse needs.

The need for asylum

Recent years have seen a rediscovery of the 'asylum' functions of the large hospital (Bachrach, 1984). The term is somewhat ambiguous (perhaps part of its attraction), but usually refers to the need for a sheltered or protective environment, as distinct from ordinary community living. Psychiatric hospital is one such environment, but 'hostel wards' whether on or off hospital sites could also be classified as taking on the function of asylum (South East Thames Regional Health Authority, 1988).

There are a range of factors that can make someone a candidate for asylum. Gudeman & Shore (1984) identified five groups of American patients requiring a "new class of facilities for the mentally ill" (p. 332), arguing that each needed different types of setting but that the facilities might reasonably be on the same campus. Wing & Furlong (1986) have argued similarly in their discussion of the proposed 'haven' on the site of Friern Hospital in north London. They identified five factors which could limit the individual's capacity to function independently: (a) risk of harm to self or others; (b) unpredictability of behaviour and liability to relapse; (c) poor motivation and capacity for self-management or performance of social roles; (d) lack of insight; (e) low public acceptability.

The present study included indices of all these factors and identified a number of overlapping subpopulations who may be eligible for 'asylum' care, including the new long-stay patients with severe problems of dangerousness or social unacceptability, those recommended for hospital placement, and others with combinations of high levels of physical disability, deficits in social functioning, or behavioural problems.

There is a problem moving from broad agreement on relevant factors to an estimate of the numbers of such people in existence in a particular district. The total may be thought of as having two components: patients already in hospital who will require long-term 'asylum' and patients likely to accumulate in coming years. Estimating the size of these components is complicated by factors such as differing mortality rates in young and old populations, the fact that some patients may require extended but not indefinite asylum care (e.g. the high proportion of the new long-stay who remain in hospital for one to three years who are subsequently discharged), and the difficulty of estimating the size of the population from which the present patients are drawn because of changes in catchment area boundaries. In addition, rates of accumulation depend upon admission and discharge policies, availability of other facilities, and variations in demography – factors which will themselves alter as community-based care develops. For all these reasons, as Wing & Furlong (1986) state, "there can be no such thing as a national or even a regional norm" (p. 452).

Wing & Furlong do suggest a 'best estimate' of 50 'high dependency' places per 100 000 population for an average district. In the current study the 'high-dependency' group may be taken to consist of those patients recommended for placements with 24-hour staffing, an assumption that produces a figure of approximately 60 places per 100 000. Of these, 40% were recommended for long-term hospital placement, giving a rate of 24 'hospital' places per 100 000, the remainder being recommended for other types of sheltered housing. The high average age of both the high-dependency group as a whole and the 'hospital placement' group in particular indicates that the number of high-dependency (and 'hospital') places required in future will be substantially lower than those needed for the present long-stay population. This is because the attrition rate will be far greater than the rate of newly accumulating patients requiring long-term care. Three ways of estimating this latter figure have been referred to: (a) the total number of newly accumulating patients with a stay of over one year, a figure of 2–4 per 100 000 per annum in the hospitals surveyed; (b) the number of newly accumulating patients with a stay of over three years, averaging 2 per 100 000 per annum; and (c) the number of newly accumulating people recommended for hospital placement, a maximum of 0.5 per 100 000 per annum. Precisely how many asylum places will be needed will depend also on what else is available. Districts will have to make calculations based on as realistic assumptions as possible.

Should the hospitals close?

The question of asylum relates to the controversy surrounding present moves towards hospital closure. On grounds of absolute need, the study shows that few patients were so disabled that they could not manage to live in the community if support was provided. Indeed, it was surprising to find that about a third of the population were thought potentially able to manage with low levels of support. Fewer patients were recommended for long-term hospital placement than in other studies (e.g. Ford *et al*, 1987; Levene *et al*, 1985), probably due to the extensive range of alternatives from which staff could select. However, the proportion recommended for 24-hour staffing was broadly comparable.

The most pressing need is therefore for highly staffed accommodation in the community. As there is currently little being provided (Audit Commission, 1986), it is reasonable to anticipate more people than necessary accumulating in hospital or prison, or becoming homeless. Whether the response to this should be to halt the hospital closure programmes is another matter: this might stifle planned developments and lead to a withdrawal of the few extra resources that are being put into services for people with long-term mental illness. Furthermore, the problems of the large geographically distant institutions have not gone away and may be getting worse. The hospital fabric, unattractive as it often is, is also exorbitantly expensive to maintain; staff recruitment is increasingly difficult and morale often low. It is all too easy to paint an idealised picture of the days of the old asylum and forget the images that emerged from the hospital inquiries (Martin, 1984). The problem of neglect of the most disadvantaged members of society, whether in hospital or in the community, seems unlikely to go away.

Another argument for halting the hospital closure programme is that the lack of replacement services is leading to an excessive burden being placed on relatives of sufferers from severe mental illness (National Schizophrenia Fellowship, 1988). However, few patients, even from the new long-stay population, were thought likely to be discharged back to the care of their families. It may therefore be misleading to suppose that if services were frozen as they are now, this would reduce family burden by preventing discharges back to unsupported families. It is more likely that early discharges from acute wards rather than long-stay hospitals are resulting in additional family burden, a probable by-product of a reduction in acute beds, increased reluctance to transfer patients from acute to long-stay wards, and the disappearance of 'medium-stay' wards. If this is so, only changes in admission and discharge policies to both acute and long-stay services, coupled with an expansion of long-stay beds, would result in more patients currently living with families being cared for in hospital. To the extent that this seems unlikely to occur, the question of the continued need for large hospitals is largely independent of the availability of adequate support to patients and relatives in the community.

Conclusions

(a) The majority of long-stay patients currently in hospital are severely disabled but could manage to live in the community with support. There is thus little long-term justification for keeping large long-stay psychiatric hospitals, assuming that provision is made in the community.

(b) In the absence of alternative provision, new long-stay patients continue to accumulate and now account for up to a third of the hospital population. They include a significant number of patients over 65 years old. Many of these are women with affective disorders; some are ageing sufferers from schizophrenia for whom community support has broken down. Their needs have as yet not been sufficiently explored and require urgent attention. In recognition of these subpopulations, the definition of 'new long-stay' should be extended to include those over 65 years old with no diagnosis of dementia and those in hospital between five and ten years.

(c) About 20% of the new long-stay population have organic diagnoses. They are often significantly more disabled than other patients. Their needs require detailed assessment.

(d) There are a small number of patients who are likely to need long-term asylum care, and this needs to be acknowledged in district plans. How many will depend both on the characteristics of the individuals *and* on the availability of alternative local provision. No definition of 'needing asylum' that depends entirely on individual characteristics should therefore be adopted.

(e) Planning on the basis of national norms is futile. Local assessments of need, taking into account the previous conclusions, are essential if resources are not to be wasted by developing inappropriate services.

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Lithium Maintenance:
1. A Standard Education Programme for Patients

MALCOLM PEET and NORMAN S. HARVEY

A videotape lecture and written hand-out containing factual information about lithium were given to 30 attenders at a lithium clinic. A further 30 patients acted as a control group and were not given the programme until later in the study. The educational programme resulted in substantial and significant increases in patient knowledge about lithium, such that knowledge increased from a baseline comparable with that of social workers to a level similar to that of community psychiatric nurses. Patients' attitudes to lithium also became more favourable after education.

In the last ten years there have been numerous studies of the effects of health education in many common conditions such as hypertension (Garrity & Garrity, 1985), and diabetes mellitus (Bush, 1987). A meta-analysis of health-education programmes (Mullen *et al*, 1985) showed that the most beneficial interventions were those providing an interactive process of learning between patients and health professionals. They also noted the importance of matching the information with its measured objectives, of tailoring the programme to the socio-educational level of the patients (Weiner & Lovitt, 1984), and of personalising treatment schedules to fit the individual routines of patients. Their overall impression was that written information on its own is of no measurable benefit to the chronically ill, and it has been shown that audiovisual instruction can be more effective in improving compliance (Stitt & Trinca, 1979).

Seventy-five per cent of relapses among lithium patients are thought to be due to poor compliance (Baastrup, 1971). Many lithium clinics provide patients with information booklets concerning their treatment (Walker, 1981). Other information-giving procedures are often elaborate, providing factual information, counselling of family members, and some supportive counselling to patients (Frank *et al*, 1985). The extent to which education about lithium plays a part in such programmes has not been described.

In our study we have provided a standard, highly specific information programme capable of precise reproduction in any other clinic or research setting, and its effects were quantified. As patient instruction on its own may have an adverse effect on attitudes to ongoing treatment (Garrity & Garrity, 1985), we also measured attitude on our Lithium Attitudes Questionnaire.

Method

Sixty consecutive attenders in remission at a lithium clinic were entered into the study over six weeks. They were divided randomly into an education group and a control group, with 30 subjects in each; all were tested individually in a quiet room with the Lithium Knowledge Test (LKT) and the Lithium Attitudes Questionnaire (LAQ). These tests were specially designed for the present study, the LKT to test knowledge essential for the safe and effective use of lithium, and the LAQ to measure attitudes opposed to lithium treatment. During the course of the study one patient from the education group dropped out and one patient died before the tests at 24 weeks, but is otherwise included in the analyses.

At their second six-weekly visit both groups were retested and then the education group was given a simple 12-minute videotaped lecture, with graphic illustrations of how lithium is used to treat affective disorders. It was designed to contain all the information required to answer the LKT and was complemented with an illustrated transcript. The programme was given to groups of up to eight patients at a time, and they were told that they would be visited at home by the authors after two weeks, so that they could discuss any of their own particular difficulties with lithium. The patients were not at that time well known to the authors, who were new to the clinic.

During the home visits the authors spent a short time answering any spontaneous questions about lithium. The patients were then given a surprise test with the LKT.

At the following six-weekly clinic, both groups were again tested with the LKT and the LAQ, and then the control group was given the information programme. They were not visited at home but had a six-week follow-up test with the LKT and LAQ at their next clinic attendance. Six weeks later still, both groups were given final long-term follow-up tests on the LKT and LAQ, 24 weeks after the start of the study.

The LKT was also given under supervision to 25 community psychiatric nurses (CPNs), 44 social workers (SWs) undergoing higher training for approval under the Mental Health Act, and 19 trainee psychiatrists, for the purpose of comparing knowledge of lithium across the groups.

Table 1
Characteristics of education and control groups (means (s.e.m.s))

	Education (n=30)[1]		Control (n=30)[2]	
Age· years	52	(2.2)	57	(2.6)
Duration of illness· years	17.8	(1.5)	18.2	(2.4)
Number of relapses	8.3	(1.7)	6.6	(0.8)
Duration of treatment. years	6.8	(0.8)	7.1	(1.1)
Daily dose of lithium: mg	1038	(52.6)	967	(58.2)
Duration of remission: years	4.5	(0.7)	4.6	(0.9)
Number of relapses on lithium	1.1	(0.4)	1.1	(0.3)

[1] Eight men, 22 women.
[2] Twelve men, 18 women.

Results

Epidemiological and clinical data for the study and control groups are very similar (Table 1). The LKT and LAQ scores showed normal distributions and were therefore analysed using parametric statistics. Student's t-tests with two-tailed P values were used throughout.

The LKT was scored by giving one point for each correct answer and removing one point for each incorrect answer, to compensate for points acquired by guessing. Incorrect answers to items which needed to be known for the physical safety of the individual on lithium were added together to form a separate hazard score (LKTH), which showed a normal distribution. The professional and patient groups were compared on both these scores.

The LKT scores obtained by the trainee psychiatrists were higher than those of CPNs, which in turn were higher than those of SWs (Fig. 1). The means (s.e.m.) were, for the trainee psychiatrists, 16.8 (0.6), the CPNs 12.6 (0.6) and the SWs 6.3 (0.5). These differences were highly significant, using a one-way analysis of variance (F ratio = 88.5, $P < 0.00005$). The mean (s.e.m.) score of the education group (5.8 (0.8)) was comparable on entering the study with that of the SWs.

The mean (s.e.m.) LKTH scores were 1.7 (0.3) for the trainee psychiatrists, 2.2 (0.2) for the CPNs, and 3.5 (0.1) for the SWs. The differences were highly significant, using a one-way analysis of variance (F ratio = 28.3, $P < 0.00005$). The education group was appreciably worse than all the professional groups, scoring 4.4 (0.2) on entering the study.

Figure 2 shows that the groups entered the study with similar LKT scores. Six weeks after receiving the information programme, the improvement in the education group's LKT scores since the onset of the study was significantly greater than that for the controls ($t = -4.86$, $P < 0.0005$). The mean (s.e.m.) LKT score (11.5 (1.0)) had reached a level comparable with that of the CPNs. Once improved, there was hardly any decline in the LKT scores of the education group during the rest of the study.

For the control group there was a slight although insignificant practice effect during the second administration of the LKT, and an obvious improvement after they received the information programme ($t = -2.95$, $P < 0.007$). This improvement was less than that of the education group, and it did not show a significant decline by the end of the study, nor did the final LKT scores differ significantly between the two groups.

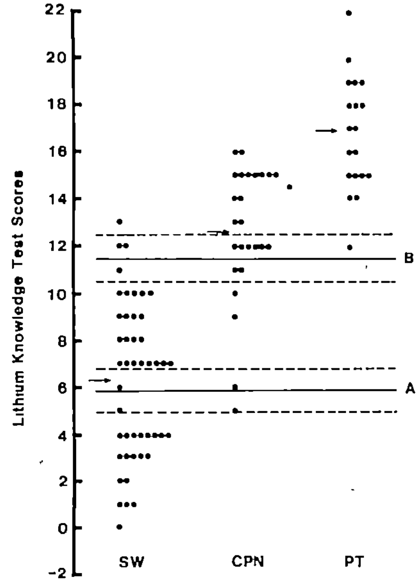

Fig. 1 The LKT scores for the three professional groups are shown, with arrows indicating means. For the education group, the means (s.e.m.) are given for the time of entry to the study (A) and for two weeks after the information programme (B) (dotted lines indicate s.e.m.).

The groups commenced with small differences in LAQ scores (Fig. 2). After the education group received the information programme, the LAQ scores improved significantly over the following six weeks ($t = 3.41$, $P < 0.003$); and they were significantly better at 12 weeks than those of the control group ($t = 2.76$, $P < 0.01$). However, this latter finding appears to be due to each group starting from a different baseline, because when the decrease was compared with changes in the control group over the same period, the decrease just failed to reach statistical significance ($t = -1.88$, $P = 0.066$). Thus the education group's improvement was not mainly due specifically to the information programme. The overall improvement in attitude by the end of the study reached significance for the groups combined ($t = 2.98$, $P < 0.005$).

To ascertain whether improved LKT scores were associated with practical benefits for the patients, the LKTH scores were examined (Fig. 3). In the education group, the mean score had dropped to half its former level six weeks after the information programme was given ($t = 4.99$, $P < 0.0005$) and it remained at this level until the end of the study, 16 weeks later. The diminution in LKTH was highly significant ($t = 4.73$, $P < 0.0005$) relative to changes in the control group. The mean (s.e.m.) LKTH score of 2.3 (0.4) after education was comparable with that of the CPNs. The controls also improved on the LKTH score after they received the programme ($t = 3.71$, $P < 0.002$).

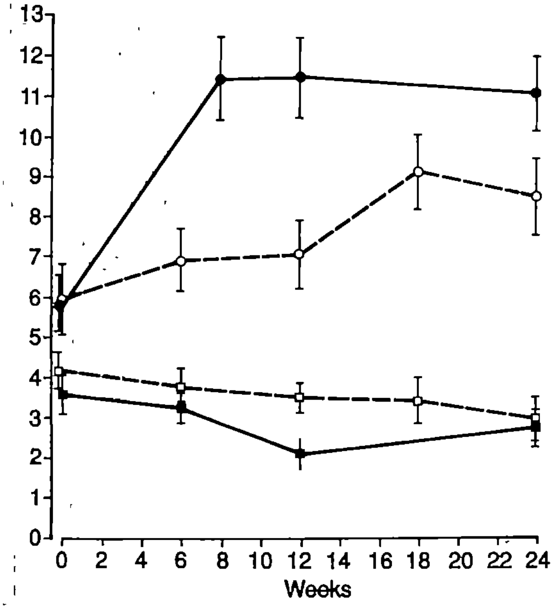

Fig. 2. Effects of the information programme (given to education group on week 6, and to control group on week 12) on LKT (●—●, education group; ○—○, control group) and LAQ scores (■—■, education group; □—□, control group). Error bars indicate s.e.m.

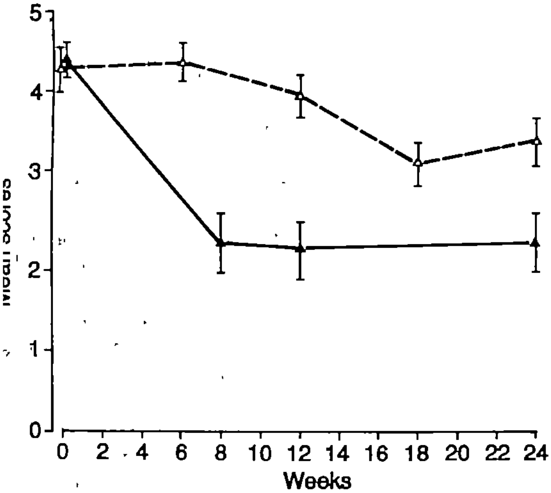

Fig. 3 Effects of the information programme on the LKT hazard score (▲—▲, education group; △—△, control group). Error bars indicate s.e.m.

Discussion

We have shown that a relatively simple educational programme, comprising a videotape, a written hand-out and a follow-up home visit, produced a substantial and sustained improvement in patient knowledge about their lithium treatment. A similar educational programme without the follow-up home visit produced a lesser, although still substantial, acquisition of knowledge about lithium. It is likely that the follow-up visit stimulated patients to revise from their information leaflets. Whether it was the videotape, the written hand-out or a combination of the two that was important for knowledge acquisition was not ascertained in our study. However, it has been previously reported that written information alone is of little benefit (Mullen *et al*, 1985).

Our data suggest that the knowledge gained by patients was not only statistically significant, but also meaningful. The LKT appears to differentiate well between professions that would be expected to have different levels of knowledge about lithium. The patients' level of knowledge was initially similar to that of SWs and rose into the range of CPNs after education. More importantly, the hazard scores of the patients, which at the outset had been higher than those for any of the professional groups tested, fell to a range comparable with that of the CPNs. Since the hazard items were related directly to the safety of lithium treatment, such as what to do if diarrhoea and vomiting occur and not to take a higher dose of lithium when feeling depressed, it would be expected that this reduction in the hazard score would lead to increased safety of lithium treatment.

We were concerned that an educational programme which included details of side-effects and toxic effects might adversely affect patient attitudes towards lithium. However, our data show that the reverse was the case, so that during the education programme there was a modest improvement in attitudes towards lithium.

The patients in our study were attending a well established lithium clinic for an average of approximately seven years. The number of misconceptions they had regarding their lithium treatment, such as the notion that they suffered from a lithium deficiency, was therefore surprising. It is clear that patients can fit very well into the routine of a lithium clinic without having any real understanding of their treatment, and that lack of knowledge is apparent only when knowledge is formally tested or when untoward events occur.

On the basis of our work, it would seem that there is a strong case for giving a structured educational programme of this type to all patients receiving lithium therapy.

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Acknowledgements and authors' details are given at the end of the following paper, pp. 203, 204.

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Lithium Maintenance: 2. Effects of Personality and Attitude on Health Information Acquisition and Compliance

NORMAN S. HARVEY and MALCOLM PEET

During a controlled education programme, the medication compliance of 60 attenders at a lithium clinic was tested using reported tablet omissions and intra-erythrocytic lithium levels. Both measures improved, along with patients' knowledge and attitude. These effects were examined and related to aspects of obsessional trait and health locus of control.

The effects of health education on patients' compliance with lithium treatment have rarely been studied, although a behavioural programme directly encouraging lithium compliance has been shown to be successful (Cochran, 1984). Reviewing a range of health-education programmes, Morris & Halperin (1979) concluded that although written information improved compliance for drugs used in the short term, this was not necessarily so in the longer term. Stitt & Trinca (1979) used a programme giving written or audiovisual information to non-psychiatric patients and, although they were unable to show a short-term improvement in knowledge, the tablet compliance of the audiovisual group improved. In view of the inconsistencies of these studies, the present work examines how a standard factual programme of audiovisual and written information, known to increase knowledge about lithium treatment (see previous paper, this issue, pp. 197–200), affects compliance and reported side-effects.

There are many individual factors that modulate acquisition of information and improved compliance during such programmes. A great deal of work exists for locus of control, and health-related information seeking (Wallston *et al*, 1976; Strickland, 1978; Toner & Manuck, 1979) although not for patients on lithium therapy. We have used the health locus in our study and we have also looked at obsessional trait, postulating that self-regulatory behaviour is important in determining patients' responses to new information. Earlier work has linked obsessionality with inflexible, perseverative thinking (Harvey, 1986). Mental rigidity is considered by Rokeach (1960) and Budner (1962) to be synonymous with intolerance for ambiguity, which Rokeach suggests is negatively related to acceptance of information.

Methods of measuring compliance vary greatly. Tests of high validity, such as an unannounced urine test, reduce the apparent effectiveness of interventive programmes intended to improve patient compliance (Mullen *et al*, 1985). Such rigorous methods have been used on a small scale in lithium patients. Schwarcz & Silbergeld (1983) used serum lithium spot checks, but most researchers use tablet counts, serum lithium levels, self-reported compliance, collateral

Table 1
Intra- and intergroup comparison of changes in compliance-related variables

	Education group ($n=29$)		Before v. after	Change in education group v. change in control group	Combined group ($n=59$)		Before v after
	Before: mean (s.e.)	After: mean (s.e.)			Before: mean (s.e.m.)	After: mean (s.e.m.)	
Lithium missed: days per 6 weeks	0.86 (0.32)	0.67 (0.23)	$P=0.07^1$	$P<0.03^2$	0.90 (0.23)	0.61 (0.24)	$P<0.02^1$
Lithium RBC levels: mmol/l	0.25 (0.02)	0.31 (0.02)	$P<0.02^3$	NS^4	0.26 (0.02)	0.30 (0.02)	$P=0.06^3$
Lithium serum levels: mmol/l	0.67 (0.05)	0.65 (0.03)	NS^3	NS^4	0.65 (0.03)	0.68 (0.03)	NS^3
Lithium ratio	0.37 (0.03)	0.47 (0.03)	$P<0.0005^3$	NS^4	0.40 (0.02)	0.44 (0.02)	NS^3
Symptom score	10.0 (1.3)	9.7 (1.3)	NS^3	NS^4	10.00 (1.2)	9.7 (1.00)	NS^3

1. Wilcoxon matched-pairs signed-ranks test (two-tailed).
2. Mann–Whitney *U* test (two-tailed).
3. Paired *t*-test (two-tailed).
4. Two-sample *t*-test (two-tailed).

reports, attendance records, clinicians' overall impression of compliance, or more often a combination of these (Connelly *et al*, 1982; Jamison & Akiskal, 1983; Cochran, 1984; Robinson *et al*, 1986).

Gengo *et al* (1980) suggested that the ratio of the concentration of lithium in red blood cells (RBC) to that in serum would be a more appropriate guide to compliance than serum lithium levels alone. This is because non-compliant patients may take lithium just before clinic attendance to simulate compliance (Schwarcz, 1980), but the low RBC level is restored less rapidly than the serum level, resulting in a low lithium ratio. However, on its own the lithium ratio would not detect tablet omissions just before testing. In this case the serum level would drop more rapidly than the RBC level, pushing up the lithium ratio. For this reason we have chosen to measure changes in compliance using serial RBC lithium levels during a fixed dosage regime.

Method

Sixty attenders at a lithium clinic entered our study, and 59 patients completed an education programme (see previous paper, this issue, pp. 197–200). During the study they were maintained on a fixed-dose lithium regime. All patients were assessed with the Lithium Knowledge Test (LKT), the Lithium Attitudes Questionnaire (LAQ), and for missed doses, blood lithium levels, and side-effects, at six-weekly test sessions over 24 weeks.

The number of missed doses patients reported was checked in private, without prior warning, with their spouses or persons with the closest involvement with them. This was done at two points in the study, and was adjusted for frequency of doses prescribed, to estimate the number of days of treatment missed in each six-week period. Serum lithium levels were measured, as well as RBC lithium levels by the indirect method (Summerton *et al*, 1989). The side-effects score was obtained from a self-report symptom check-list (Ghose, 1977).

All patients were tested once with the Health Locus of Control (Toner & Manuck, 1979) and with the Leyton Obsessional Inventory (Cooper, 1970). Nine of Cooper's original subscores were chosen as relevant to the present study, as well as the aggressiveness subscore proposed by Kendell & Discipio (1970) in their study of obsessionality and affective disorders. The original subscores chosen are as follows: checking, order and routine, repetition, overconscientiousness and lack of satisfaction, indecision, rigidity, health, regularity, and punctuality.

The present study compares contemporaneous changes in the education and control groups when only the former had received the programme. It then combines the changes occurring in both groups, just before and six weeks after each had received the programme. This is to provide sufficient numbers of patients to examine variables associated with improved knowledge of and attitude to lithium.

Results

Independent observer-based estimates of missed doses were obtained in 49 cases. There were two refusals and eight patients lived alone without regular support. These rough and insensitive estimates correlated with patients' estimates ($r=0.60$, $P<0.0005$), but tended to underestimate the number of missed doses.

Table 1 shows changes in the education group during the six weeks after they received their programme (see also Fig. 1a). There was a reduction in doses missed, but this did not quite reach significance (using non-parametric statistics owing to its markedly skewed distribution). However, RBC lithium levels and the lithium ratio increased significantly without a corresponding increase in serum lithium (Table 1), as was expected for improved medium-term compliance.

When these changes were compared with those for the control group during the same period (Fig. 1a,b), only the reduction in missed doses was of significance (Table 1). Thus the information programme was associated with a significant improvement in reported tablet compliance. However, the accompanying increases in blood lithium levels, which we

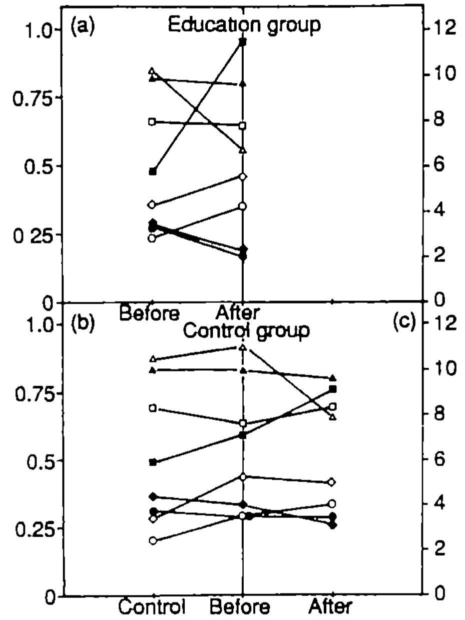

Fig. 1 Changes in knowledge, attitude and compliance-related variables during the information programme (Δ lithium missed, ○ RBC lithium, □ serum lithium, ◇ lithium ratio, ■ knowledge score, ◆ hazard score, ● attitude score, ▲ symptom score).

consider more objective measures of compliance, were not specifically associated with the information programme.

Results from the education and control groups were combined for changes in reported tablet compliance, blood lithium readings, and side-effects (Table 1), for the two six-week periods after their respective programmes (Fig. 1a,c). There was a significant reduction in doses missed and an increase in RBC lithium which just failed to reach significance. There was no accompanying increase in reported side-effects.

The relations among improvements in the above compliance-related variables and improvements in knowledge and attitude were explored for the combined group. Improved attitude was significantly correlated with reported reduction in missed doses ($r=0.332$, $P<0.006$). The relative validity of the reported reduction is substantiated by the parallel improvement in RBC lithium levels ($r=2.43$, $P<0.04$) and lithium ratio ($r=0.248$, $P<0.03$). Increased lithium knowledge was also found to be associated with improved tablet compliance ($P<0.05$), and it correlated negatively with serum lithium levels ($r=-0.291$, $P<0.02$). The latter finding is compatible with patients less often needing to take covert doses on the morning of the test to simulate compliance.

Improvement in the knowledge score, improved attitude, and reported reduction in missed doses were examined for associations with the selected Leyton subscores. Owing to its practical value, the hazard score of the LKT was included in the analysis. The following relationships emerged. Overconscientousness and lack of satisfaction was associated with improvement in the hazard score ($r=0.265$, $P<0.03$). Those with obsessional health concerns acquired less information from the programme ($r=-0.314$, $P<0.01$), and showed somewhat less improvement in their hazard scores ($r=-0.211$, $P<0.06$). Obsessional aggressiveness was associated with less improvement in attitude ($r=-0.299$, $P<0.02$), and less reduction in doses missed ($r=-0.238$, $P<0.04$). Rigidity was associated with less improvement in doses missed ($r=-0.227$, $P<0.05$). Since the education group had more opportunities to increase their level of knowledge acquisition, during the domiciliary visit, an association was sought in this group between knowledge acquisition from the programme and health locus of control, but none was found ($r=0.04$, $P=0.4$).

Discussion

Improvement in compliance was discernible after this brief factual information programme. However, both the education group and the control group showed increases in their RBC lithium levels and lithium ratios, without any accompanying increase in their serium lithium levels. This strongly suggests improved tablet compliance in both groups. It therefore appears that the information programme itself, although not primarily responsible for this biochemical evidence of improved compliance, created an anticipatory interest in both groups which exerted a general effect on their compliance. Biochemical evidence of improvement in compliance reached significance only in the education group, no doubt because of the effect of receiving a domiciliary visit in addition to the videotape and hand-out.

The increase in RBC lithium in the education group was accompanied by a significant reduction in reported doses missed. However, there was no similar reduction in the control group and yet they showed biochemical evidence of improvement. Perhaps the education group gave a more accurate history of omissions owing to their slightly greater involvement in the programme, although we were unaware of such effects during the study. Also, recollection of omissions may be lower for those forgetting their treatment, and others in their immediate environment would not always be aware of the problem. Often it seemed that the strong relationship between patients' and informants' assessments of missed doses must be largely due to the informants' long-term experience of the average number of doses missed, rather than a constantly updated appraisal.

After both groups had received the programme, improved attitude to lithium treatment on the LAQ, in the combined groups, was related to biochemical evidence of improved compliance. No such biochemical evidence occurred in relation to improved lithium knowledge, which was nonetheless strongly associated with reported improvement in doses missed. This reported improvement may have occurred because, with greater knowledge, social desirability of compliance increased, biasing patients' reports. Thus improved lithium attitude appears to be the more reliable factor relating to improved compliance, supported with biochemical evidence.

It is of interest that health locus of control did not relate to knowledge acquisition in the education group. Our impression was that patients did not greatly increase their knowledge of lithium during the home visits, but that their clearly greater knowledge acquisition was probably due to some preparation for or anticipation of the visits. In either case health locus should have determined knowledge acquisition. If the locus was internal, their sense of responsibility for their health would have led them to revise more than those whose locus was external. It was not expected that this plausible hypothesis would be unsupported in the present study.

We have found, in work to be published elsewhere, that obsessional characteristics were no more prevalent among the lithium patients in the present study than in the general population. Thus there is no suggestion that the group as a whole showed pathological obsessionality. Within the group, however, certain specific obsessional characteristics were of relevance to knowledge acquisition, changed attitudes and compliance. Overconscientious patients who lacked satisfaction significantly improved their knowledge about the potential dangers of lithium treatment.

Obsessional health preoccupation was associated with lower knowledge acquisition, and also with somewhat less improvement in hazard scores. These patients were not actively depressed but admitted to having been preoccupied with their bowels or having given thought to the possibility of cancer. Thus they show what could be regarded as the more hypochondriacal aspects of obsessional thought. They had no greater knowledge of lithium at the onset of the study, and absorbed less information from the programme. It may be that their health concern reflects an awareness of their own difficulties in grasping and absorbing health information.

Improved attitude and tablet compliance were less likely to occur in patients scoring higher for aggressive obsessional ruminations. Their knowledge acquisition was no lower than for the rest of the patients, so it seems that personality rather than lack of information may have played a part in their poor compliance response. Tablet compliance was also less likely to improve in patients showing obsessional rigidity. This latter observation adds behavioural relevance to previous neurocognitive work on obsessional rigidity (Harvey, 1986).

In conclusion, our information programme was presented in a standard form which is capable of duplication in most lithium clinics. It appears to promote a range of improvements in lithium patients without increasing reported side-effects, and allows the study of factors modulating these improvements.

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Copies of the video-tape and information hand-out, and the LKT and LAQ, are available as a package from Sheffield University Television, Sheffield S10 2TN.

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Borderline and Schizotypal Disorders in Children and Adolescents

MARTIJN MEIJER and PHILIP D. A. TREFFERS

Until recently, research on borderline disorder in children has sought the common denominator of the symptoms. In recent years there have been attempts to circumscribe the definition with the help of DSM–III criteria and the DIB. This approach appears fruitful. The scanty data on schizotypal children suggest that the validity of this diagnosis in childhood should be investigated. In adolescence it is possible to discern those with borderline and schizotypal disorders whose symptoms meet both DIB and DSM–III–R criteria respectively. No data exist, however, concerning the predictive validity of such disorders in adolescents. Classification on an empirical basis is advocated in order to refine the diagnosis of these and related disorders in children and adolescents.

In most child and adolescent psychiatric handbooks only a modest paragraph is devoted to borderline disorders. Some textbooks deal exclusively with borderline disorders in children (e.g. Noshspitz, 1979); others mention only those in adolescents (e.g. Rutter & Hersov, 1985). It is often stated that the disorder is not uncommon, and certainly occurs more frequently than autism, a disorder described extensively in almost every manual.

Evidence is growing that it may be crucial for the planning of a treatment programme for disturbed youngsters to recognise this disorder. The suicide rate in depressed adolescents is elevated when they exhibit a borderline disorder as well (Friedman *et al*, 1983). Behavioural therapy with conduct-disordered adolescents is complicated by a coexisting borderline personality disorder (Bartels, 1986). Such a disorder in adolescents alerts the clinician to severe acting out, unmanageable regression and countertransference in the future (Masterson, 1985; Lewis, 1986). In hospital settings treatment of these adolescents may require strict limit setting (Steinberg, 1983).

In the psychiatric treatment of children, too, it is important to recognise borderline disorder as soon as possible. An adequate treatment programme will help to stimulate the healthy aspects of their functioning and to diminish the harmful influence of this disorder, which may eventually result in low self-esteem (Lewis, 1986) and in paranoid defences.

There has been a lack of clarity in defining borderline disorders in children and adolescents, which emerges in different ways. Often the discussion focuses on the deficiencies of different aspects of the diagnostic validity. Thus, for example, the level on which the symptoms are described in various publications differs sharply. Some authors reproduce tangible data, others avail themselves of metapsychological terminology, without mentioning what observable behaviour these formulations refer to (Shapiro, 1983).

Moreover, defining borderline disorders of children and adolescents is particularly complex because the question has not been answered as to whether it is the same disorder in both groups. This is partially because descriptions of borderline children come primarily from child psychiatric literature, while those relating to adolescents are mainly derived from the field of adult psychiatry. In the literature on adolescents little connection is made with data from child psychiatric literature, and vice versa.

The concept of borderline in adult psychiatric literature has taken on a more limited meaning since the introduction of DSM–III (American Psychiatric Association, 1980) in which, in comparison with only a passing mention in DSM–II, the concept received ample attention. Spitzer *et al*'s (1979) initial suggestion that there were two borderline categories – one group of patients related to schizophrenics genetically and in terms of symptoms ('borderline schizophrenia'), and another group whose personality is characterised by instability and vulnerability in accordance with the 'borderline personality organization' of Kernberg (1975) – was confirmed in a survey of 808 American psychiatrists. Thus, in DSM–III they differentiated between schizotypal personality disorder (SPD) and borderline personality disorder (BPD). Criteria for the diagnosis BPD were derived largely from the conclusions of Gunderson & Singer's (1975) extensive review of the literature, and these criteria in DSM–III–R (American Psychiatric Association, 1987) were little changed (see also Widiger *et al*, 1988).

Several other diagnostic instruments have been developed, with criteria related to those of DSM–III, to identify patients with BPD. The best of these, the Diagnostic Interview for Borderline Patients (DIB; Gunderson *et al*, 1981), is a semistructured interview with criteria that are largely the same as those of DSM–III; although it also stresses the importance of short-lived psychotic manifestations.

Experience with DSM-III-R criteria for children and adolescents is still limited.

In a review on borderline personality of adults, Tarnopolsky & Berelowitz (1987) concluded that: "What before was a vague, mythical or muddled concept has achieved in many ways the significance of other commonly used psychiatric disorders." However, data on descriptive and predictive validity for the borderline concept in children and adolescents are still so scarce that empirically based pronouncements such as these cannot be made.

In this article we chart the available data on borderline and schizotypal pathology in children and adolescents. We exclude data concerning treatment, and we formulate some conclusions regarding the usefulness of these diagnostic categories in children and adolescents. Moreover, we formulate recommendations with regard to research in this area.

In children and adolescents, it is permissible to diagnose personality disorders when they show persistently, over a long period of time, the essential characteristics of any personality disorder described in DSM-III-R (Lewis, 1986). Children and adolescents with long-lasting borderline or schizotypal symptoms surely exist, but in individual cases, it is difficult or even impossible to predict which symptoms will persist. Data on the vicissitudes of these disorders in children and adolescents are scarce. For that reason we prefer to speak of 'borderline disorder' and of 'schizotypal disorder' in these age groups, although what we mean by these concepts is the presence at any given moment of symptoms that are described in DSM-III-R under BPD and SPD, respectively.

Borderline disorders in children

Symptoms

Until the end of the 1970s, we understood borderline children to be those whose symptoms – certainly at first glance – were extremely heterogeneous. The criteria used in diagnosis were so faulty that reliable data on prevalence were not, nor are yet, available. In the early 1980s, however, Vela *et al* (1980, 1983) listed and grouped the symptoms described in seven important publications on borderline children and found agreement among six of the authors with regard to six of the 19 groups of symptoms (see Table 1). Furthermore, Bemporad *et al* (1982) inventoried the symptoms of 24 latency-age patients diagnosed as borderline. The areas they indicated as characteristic of borderline children are reasonably similar to those described by Vela *et al* (1983) (see Table 1).

Table 1
Borderline symptoms in children

Criteria of Vela *et al* (1983)	Criteria of Bemporad *et al* (1981)
Disturbed interpersonal relationships	Fluctuation in functioning
Disturbances in the sense of reality	Nature and extent of anxiety
Excessive, intense anxiety	Disturbances of thought content and processes
Excessive and severe impulsive behaviour resulting from minimal provocation or frustration	Disturbed relationship to others
'Neurotic-like' symptoms	Lack of control
Uneven or distorted development	

From a theoretical viewpoint it is remarkable that the researchers repeatedly attempted to uncover the common denominator of the heterogeneous symptoms rather than asking themselves if there were perhaps more than one group. This was probably because borderline children were designated as such predominantly by child psychiatrists with a psychoanalytic orientation. The functioning of borderline children, especially in the area of object relationships, was thus approached from one theoretical angle, in which the symptoms were grouped together and interpreted as an arrest in development on the level of separation-individuation (e.g. Chethik, 1979). By placing the accent on abstract, metapsychological terms, the divergence among characteristics of these children on a phenomenological plane was concealed. Pine (1974, 1986) for example, states that: "... the concept borderline is more like the concept 'transference' or 'defense' or 'regression'. While we use these terms so often that we may think of them as having thing-quality, they are not things; they are terms that serve communication through consensus. Thus the term borderline is a concept that means only what we say it means...".

In the same period, authors began to wonder if it were possible to classify borderline children with criteria of DSM-III. Bradley (1979), in her research on the aetiology of borderline symptoms in adolescents and children, employed the criteria developed by Gunderson & Singer (1975) (forerunners of DSM-III criteria) for diagnosing borderline adults. In a later survey (Bradley, 1981) of American child and adolescent psychiatrists, she found that a large number of these considered that the 'adult criteria' of Gunderson & Singer were applicable to adolescents and – to a lesser degree – to children. With regard to the children these criteria applied, for instance, to "loose thinking in unstructured situations" and "relationships that vacillate between transparent superficiality and intense dependency."

Also Vela *et al* (1983), who it is true attempted to ascertain the common characteristics of borderline children, tested their formulated common characteristics against the DSM–III criteria. They concluded that these characteristics agreed with the criteria for SPD (in adults) and that considerable overlap exists with the criteria for BPD (in adults) and pervasive developmental disorder.

Petti & Law (1982), the first researchers to carry out small-scale empirical research among children in this area, tested the data from 10 children (aged 6–12 years) in whom borderline psychotic features were diagnosed against a number of DSM–III criteria: SPD, BPD, and pervasive developmental disorder. The data on five of the ten children satisfied the criteria for SPD; the data on the other five children satisfied the criteria for BPD; and in both groups two children met the criteria for pervasive developmental disorder. The most distinctive features of the schizotypal children were magical thinking with bizarre fantasies and preoccupations, odd speech, and inadequate rapport. The most distinctive features of the borderline children were unstable and intense relationships; inappropriate, intense or lack of control over anger; affective instability; and physically self-damaging acts.

On the other hand Gualtieri *et al* (1983) found that none of their 16 children (aged 6–13 years) who were diagnosed as borderline satisfied the 'adult' DSM–III criteria. Greenman *et al* (1986) retrospectively investigated the material relating to 86 children aged 6–12 years using a version of the DIB. Of these children, 57 had been admitted because of suicidal tendencies and 29 showed serious psychopathology of a non-psychotic nature. Twenty-seven children (31%) met DIB criteria for BPD. The authors concluded that, on the one hand, there was a certain conformity with the syndrome as defined according to adult criteria; on the other hand, however, the criteria for adult BPD encompassed no clear, significant syndrome in this group of children. Greenman *et al* (1986) indicated that it is important in future research to include the presence of anxiety and fearfulness or phobic behaviour, which in their study had not been the case. Unfortunately, the analysis of the data limited itself to borderline diagnosis and the material was not examined for schizotypal characteristics.

Aetiology

With regard to the aetiology of borderline disorder in children a number of divergent conceptions exist. In the psychodynamically orientated literature the aetiological emphasis is usually placed on the experiences of very early traumas: "It is the child's experience of the stimulus which must be emphasized; the stimulus itself may not be externally identifiable as traumatic" (Pine, 1986). This gives rise to developmental defects. For instance, the development of trust in the caretaker is impaired and the evolution of panic anxiety to signal anxiety fails to occur. This leads to the development of ultimately maladaptive survival mechanisms for coping (Pine, 1986). This opinion is supported by the reported occurrence of physical abuse and neglect in families where borderline children grow up (Greenman *et al*, 1986; Bemporad *et al*, 1982).

Results supporting early separations as a causative factor in borderline pathology are contradictory: Bradley (1979) found them but Greenman *et al* (1986) did not in children diagnosed according to the criteria of Gunderson & Singer (1975) and the DIB respectively. Other investigators looked for organic impairment as a causative factor. Bemporad *et al* (1982), in children diagnosed as borderline with their own criteria (Table 1), frequently found poor coordination, perceptual motor difficulties, hyperactivity, and non-specific electroencephalogram tracings. Cohen *et al* (1983) point to the overlap between borderline disorder and attention-deficit disorder (ADD) and consider "that the ADD and borderline syndromes might represent, at least in part, similar clinical entities with some common underlying biological dysfunctions". However, Greenman *et al* (1986) did not encounter more frequent neurological deviations in borderline children than in their control group.

We previously stated that the criteria of Vela *et al* (1983) overlap considerably with the criteria of pervasive developmental disorder and we mentioned empirical research in this area (Petti & Law, 1982). Borderline disorder of children might be placed within the DSM–III–R category "pervasive developmental disorder, not otherwise specified", but because of the wide definition of this category, diagnostic clarity would not be furthered in this way.

In clinical discussions the opinion is heard that autistic disorders and borderline disorder constitute a continuum. This would imply a constitutional aetiology of borderline disorder, including the same defects that are supposed to underlie autism (for the aetiology of autism, see e.g. Rutter & Schopler, 1987). However, there is no empirical research to support this opinion.

Follow-up studies

We are aware of only one follow-up study of a small group of borderline children, on whom more or less objective criteria were used in diagnosis. Kestenbaum (1983), in a 10- to 25-year follow-up of seven children who in the latency age were diagnosed as borderline, found that in all cases the children's symptoms satisfied at least five of the six consensus criteria formulated by Vela *et al* (1983). The classification of psychiatric problems in the adult years was very divergent (schizophrenia, schizotypal personality disorder, schizoid personality disorder, schizoaffective disorder, bipolar disorder, borderline personality disorder, and anxiety neurosis).

Borderline disorders in adolescents

Symptoms

A fairly extensive literature on borderline pathology in adolescents also exists, generally in the psychoanalytic tradition; in many respects it derives from and builds upon work with adult borderline patients (e.g. see Kernberg, 1975). Data concerning young adolescents are scarce. Meissner (1984), for example, states that the borderline diagnosis in early adolescence is vague and indistinct. In his opinion the symptoms later in adolescence are progressively more similar to those of adult borderline patients. According to Rinsley (1980) the borderline pathology at the onset of adolescence borders on psychosis, while the symptoms towards the end of adolescence resemble those of adult borderline patients not characterised by (longer-term) psychotic symptoms.

Masterson (1985), who in particular applied himself to the diagnosis and treatment of borderline pathology in adolescents, also mentions the possibility of transient psychotic episodes, notably in the reaction to separation. He does not consider this as characteristic of the borderline adolescent, and establishes acting out – in the broad sense – as central to the symptoms of these adolescents. Under symptoms he lists boredom, restlessness, difficulty in concentrating in school, hypochondriasis, excessive activity, and forms of antisocial behaviour. He also mentions passive–aggressive, obsessive–compulsive and schizoid symptoms, anorexia nervosa and flight in this connection. In his opinion a clinging, dependent relationship with an adult sexual partner is also typical.

Only a few studies of adolescents used DSM–III criteria or the DIB. McManus *et al* (1984*a*), in a group of 71 severely delinquent adolescents, found 26 cases of borderline personality according to DSM–III criteria, of whom 73% satisfied DIB criteria as well. From this investigation a picture emerges of the delinquent borderline adolescent as one ". . . who is superficially socially appropriate, though unable to produce any sustained achievement in work or school, is affectively labile, self-mutilative, prone to brief episodes of paranoid ideation and likely to be involved in relationships where dependency needs are great, but denied and where hostility and instability are prominent". Similar results were obtained by McManus *et al* (1984*b*) when they applied DIB and DSM–III criteria to 48 adolescents admitted to hospital.

Yanchyshyn *et al* (1986) tested both the DIB and DSM criteria on 44 clinically admitted adolescents aged 13–18 years. The DIB was carried out at admission and led to borderline diagnoses in 12 adolescents. At discharge (on average after three months) DSM–III diagnoses were made. Of the 12 DIB positives, eight received the DSM diagnoses of BPD at discharge. In addition, there were two other patients diagnosed as BPD at discharge who had been DIB negative upon admittance.

Aetiology

There is virtually no available empirical research on the aetiology of borderline disorders in adolescents. The literature on adults with a borderline disorder gives no indication of a genetically determined predisposition (Pope *et al*, 1983; Torgerson, 1984).

Drawing on his clinical experience, Masterson (1985) places the emphasis on traumatic experiences in the separation–individuation phase which lead, among other things, to such adolescents not being able to tolerate separations. In the previous histories of these adolescents Masterson identifies prolonged dependency, passivity, heightened fears of separation and phobias. Frustration tolerance, impulse control, and reality perception are poor. There are disciplinary problems in upbringing at home and at school from a young age. Interaction with peers is difficult. During childhood there are often somatic complaints. Whenever they are confronted with a loss (divorce of parents, departure from home of an older sibling) borderline adolescents react in a manner akin to the despair described by Bowlby (1969) of two-year-old children separated for too long a time from their mothers. When this 'abandonment depression' threatens to break through, the adolescent attempts to ward off the unbearably depressive feelings by acting-out behaviour.

We find related themes in Gunderson (1984), whose name is associated above all with empirical research on adult borderline patients. Gunderson links the sharply alternating symptoms that a borderline patient exhibits over the course of time to the presence or absence of a significant object and to the extent to which the patient feels sustained or frustrated by this object. Other authors have also emphasised that borderline adolescents, in the course of their development, can have tremendous problems with unavoidable separations such as going to nursery school for the first time or leaving the parental home (Soloff & Millward, 1983). Walsh (1977) determined that in a group of 14 borderline young adults the inception of the symptoms (including many suicide attempts) was closely related in 13 of them to an attempt to break loose from the parental home.

Scarce systematic studies of the way borderline adolescents are brought up reveal in most cases a lack of involvement of the parents with their children, but also open hostility, abuse and sexual maltreatment (e.g. Walsh, 1977; Gartner & Gartner, 1988; Zanarini *et al*, 1989). These families are often found to be conflict-ridden, or broken up by divorce or by admission to hospital of one of the parents (Walsh, 1977). Gunderson *et al* (1980) found that characteristics of those families include a poor enforcement of rules and the denial of the pathology of the child. Masterson's (1985) assertion that borderline adolescents always have one or two borderline parents is not empirically substantiated, although some authors report severe psychopathology of varying origin in many parents of borderline adolescents (Gunderson *et al*, 1980).

Follow-up studies

Follow-up studies up to adulthood of borderline adolescents diagnosed according to DSM–III or DIB are not available. Personality disturbances in adolescents are generally regarded as preliminary stages of affective disorders in adulthood (Gosset *et al*, 1983), but also of other pathology. Aarkrog (1981) examined 50 adolescents admitted to hospital with borderline diagnosis and retrospectively investigated their symptoms at a younger age. A follow-up also took place five years after discharge, at around age 20. Unfortunately, the (Danish) classification used does not fit the DSM–III very well. The disturbances of the adolescents in this group were sometimes manifest only in adolescence and showed some signs of improvement in young adulthood.

Masterson & Costello (1980) carried out a follow-up study of 31 adolescents treated clinically for borderline disorder who satisfied Masterson's criteria. Of these, 20 reacted well to treatment, and in 18 of them a favourable result was maintained at follow-up (on average after four years, at age 20 years). Ransohoff (1978) followed up seven adolescents, who at around age 15 years had undergone several years of psychotherapy, until they were on average age 26 years. These adolescents fit the picture of the borderline adolescent drawn by Masterson. The pathology was usually still present in the follow-up, but in a number of cases the ego functions had developed in an adult direction so that these patients were better able to control their regressive behaviour.

Long-term follow-up of young adult borderline patients reveals a fairly good prognosis in the fourth decade of life, except for about 10% of the patients who commit suicide (Stone *et al*, 1987).

Schizotypal disorder in children and adolescents

Schizotypal personality disorder was introduced in DSM–III along with the BPD. The characteristics of SPD were largely derived from the research of Kety *et al* (1968) with biological relatives of adopted schizophrenics.

Kendler (1985) reports that the current concept of SPD originates from two traditions. On the one hand, in the 'familiar' tradition (e.g. Kraepelin, 1913; Bleuler, 1911; Kretschmer, 1921; Kallmann, 1938) personality disorders were described that had been found in abnormal, but non-psychotic, family members of schizophrenic patients. On the other hand there was the 'clinical' tradition (Hoch & Polatin, 1949; Deutsch, 1942; Zilboorg, 1941; Rado, 1953). The terms 'pseudoneurotic schizophrenia' and 'ambulatory schizophrenia', among others, originate with these authors.

The criteria for SPD in DSM–III can be taken to be the common denominator of the symptoms mentioned by these two groups of authors, supplemented with a number of other symptoms (such as ideas of reference and illusions). The SPD criteria were in some way altered in DSM–III–R. The shaky status of this classification was clearly illustrated in recent research by Morey (1988). On the basis of a survey among 170 psychiatrists and psychologists he calculated that half the patients who according to DSM–III should be classified as schizotypal did not meet DSM–III–R criteria for the 'same' disturbance. Historically, it is understandable that in the professional literature far less attention is given to SPD than to BPD (Blashfield & McElroy, 1987). The former often involves individuals who themselves do not seek help but who are encountered during investigation of families of schizophrenic patients.

Schizotypal disorders in children

The classification SPD in children has received almost no attention in the professional literature.

An exception is the article of Nagy & Szatmari (1986) who turned to the literature on SPD itself, the schizoid disturbance in childhood, the syndrome of Asperger, and the 'autistic psychopathy', since the descriptions of the symptoms of children with these disorders in many ways coincided with the DSM-III criteria for SPD. Symptoms common to all disorders were the extreme degree of social isolation, an impaired capacity for empathy, a deficit in understanding non-verbal behaviour, and abnormalities of speech, thought processes, and communicative ability.

The researchers active in this area approach the problem from two strongly divergent angles. On the one side are those who make the connection with infantile autism, which is fully justified by the symptoms. On the other side are those who place SPD squarely in the schizophrenic spectrum because children with this disorder are often described in the family studies of schizophrenics. Consequently, it is entirely unclear, as Nagy & Szatmari (1986) stated, whether in children SPD is a matter of a mild form of infantile autism, a genetic variation of schizophrenia, or a totally 'independent' disorder. To obtain greater insight into this matter, Nagy & Szatmari investigated the clinical records of 20 children and adolescents who were selected on the presence of at least two of the symptoms that DSM-III lists for SPD. None of the children met the (DSM-III) criteria for infantile autism, whereas 18 of them satisfied three more criteria for SPD and could be diagnosed as having pervasive developmental disorder as well (if the age criterion of 30 months was not used). This investigation shows clearly that it makes sense to examine further the diagnosis of SPD in childhood: this appears to involve a relatively large number of children, all of whom showed serious disturbances at an early age. In another study Asarnow & Ben-Meir (1988) found that children with a SPD showed a course that ran parallel to that of schizophrenic children, a fact that was already established for adults by McGlashan (1986).

Schizotypal disorder in adolescents

We are not aware of research directed at schizotypal disorders in adolescents. In the older professional literature the 'diagnostic predecessors' of this classification are sometimes mentioned in passing (Deutsch, 1942; Rado, 1953; Hoch *et al*, 1962). SPD in adolescence is sometimes mentioned in prospective studies describing the prognosis of children and adolescents considered to have an increased likelihood of schizophrenia because of a schizophrenic parent. In five out of 12 such children 'at risk' Fish (1987) observed the development of a SPD in adolescence. These five children had showed a retardation of bodily growth and motor development in the first years of life, and feelings of depression and loneliness in their latency years.

No follow-up studies of adolescent SPD are known to us. In adults with SPD, the picture through the years remains fairly stable. A small percentage becomes chronically schizophrenic and another small group shows some improvement (e.g. Hoch *et al*, 1962; Plakun *et al*, 1985; McGlashan, 1986).

Discussion

We can as yet infer only a number of preliminary conclusions from the scarce empirical data on borderline symptoms in children. There are strong indications that already in the childhood years it is possible to identify children whose symptoms are similar to those of 'adult' BPD. The question of whether these symptoms imply a homogeneous diagnostic category is still unanswered. The validity of the diagnosis of borderline disorder in childhood has no more than a face validity: the descriptive validity has not been sufficiently established; and the predictive, construct and concurrent validity of the diagnosis have not been established at all. Data available so far suggest an aetiological heterogeneity. Divergent views are held as to the nature of constitutional deficits underlying childhood borderline disorder: should we think of a 'mild' form of autism, or do we have to consider less specific mechanisms, comparable with dysfunctions included in the concept of attention deficit disorder? So far, the view that environmental influences (abuse, neglect) contribute to this kind of pathology has received most empirical support. It is unknown whether extreme environmental adversity may suffice to produce the disorder in children without a constitutional deficit.

With regard to the borderline diagnosis in adolescents the situation is not much better, except for the descriptive validity which was examined a few times with the DIB. Surely older adolescents may display personality disorders that are indistinguishable from those of adults. However, it is also true that in the case of adolescents, satisfying the criteria for a personality disorder is not the same as being affected with an immutably anchored personality pathology. It is probable that in a number of adolescents the borderline symptoms are only manifested in, and are restricted to, adolescence because it is precisely this phase of life which places insuperable demands on individuals who cannot tolerate separation. There are no data which might indicate factors determining which adolescents remain borderline.

Empirical data on SPD in children and adolescents are even more scanty. From available data it is possible to distinguish symptoms in children and adolescents that are similar to those of 'adult' schizotypal disorder. SPD in children and adolescents probably has a heterogeneous aetiology. Connections have been suspected, for instance, both with autism and schizophrenia. Research will have to demonstrate if it is possible to 'refine' the classification of the symptoms of borderline and schizotypal children and adolescents. Greenman *et al* (1986) have indicated that one can use criteria here for 'adult' borderline disorder on the one hand and data from the literature on borderline children on the other. In a similar manner one can investigate to what extent a 'counterpart' to schizotypal personality disorder in childhood can be differentiated. That children are in a developmental state obviously complicates the formulation of criteria pertaining to childhood. It is partly for this reason – even with a carefully developed prototypal typology (Clarkin *et al*, 1983) – that by no means all children will be classifiable. A refining of the classification is a prerequisite to further research on the aetiology, progress, aspects of treatment, and prognosis.

An attempt to improve the classification, based on DSM-III-R and 'garnished' with items which may or may not be specific to childhood, will prove to be insufficient. Although one will be able to classify the symptoms of a number of children in this way, the diagnostic validity of the categories will be limited and the symptoms of countless other children will be unclassifiable.

To improve the classification it is preferable to use the empirically based classification method (e.g. see Siegel *et al*, 1986). Ideally, one will be able to classify actual behaviour and to identify homogeneous groups of children who, for instance, display a certain degree of similarity with regard to a number of pre- and perinatal data, genetic data, development of functions, symptoms in varying stages of development, and progress. An approach of this kind will not only lead to a better understanding of borderline and schizotypal children, but is also essential for obtaining insight into these problems in adolescents.

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Behaviour Disorder in Pre-school Children in Hong Kong
A Two-Stage Epidemiological Study

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A representative sample of 855 Hong-Kong Chinese children aged 36–48 months were assessed using the BSQ and the PBCL. Good reliability for both instruments were found. For the BSQ and PBCL, 12.75% and 27.6% were above the cut-off points of 10+ and 12+ respectively and 5.9% were above both cut-off points. In the second stage, 234 subjects were recruited by stratified random sampling according to the results of the screening stage. A clinician interviewed the parent, child and teacher before making a diagnosis. The prevalence of behaviour disorder was: nil, 53.7%; dubious, 23.1%; mild, 18.0%; moderate, 4.5%; and severe, 0.7%. There were significantly more boys in the categories mild, moderate and severe.

Difficulties may arise in many areas such as the control of aggression, sleep, sphincter control, peer relationships and the development of a balance between dependence and independence. Minor or transient difficulties are common in young children and should not be regarded as a disorder. However, some children show multiple problems in their behaviour which are severe and persistent enough to cause a handicap in their development and emotional suffering in both their parents and themselves. This kind of problem is referred to as behaviour disorder.

The predictive value of behaviour disorder in pre-school children is important to consider. If it is just a transient, self-remitting phenomenon, then perhaps it would be wrong to put a label on it. However, there is good evidence from group-comparison follow-up studies that behaviour disorders in pre-school children do not usually pass away spontaneously (Coleman *et al*, 1977; Abe *et al*, 1982; Richman *et al*, 1982; Baker *et al*, 1983; Lerner *et al*, 1985). The severity of the problem, as well as the type of problem such as hyperactivity or extreme shyness, are important determinants of future outcome (Fagot, 1984; Campbell *et al*, 1986; Kagan *et al*, 1988).

There have been several studies on the prevalence of behaviour disorder in children under five years old, most of which were one-stage cross-sectional studies, employing a questionnaire or an interview schedule. The Behaviour Screening Questionnaire (BSQ; Richman & Graham, 1971), a semistructured interview administered to a parent, was by far the most common instrument used and the results, which found prevalences ranging from 11.3% to 15.5%, were surprisingly consistent considering that the studies were carried out in different countries or places (Richman *et al*, 1975; Earls & Richman, 1980; Wolkind & Kruk, 1985; Cornely & Bromet, 1986).

In studies based only on mothers' assessments of their children's behaviour, the proportion of mothers who felt their children had definite problems in behaviour was found to be 22% ($n = 132$) by Jenkins *et al* (1980) but only 1.6% by Campbell & Breaux (1983). Thus, there seem to be no consistent findings if one relies on mothers' perception rather than on detailed questioning on symptoms.

Several studies have used a teacher's questionnaire to assess the problem. McQuire & Richman (1986*b*), using the Pre-school Behaviour Checklist (PBCL), an instrument developed by themselves (McQuire & Richman, 1986*a*), identified the proportion of children with behaviour problems to be 3.3% in play schools, 10.8% in nursery schools, and 34.9% in day nurseries. This demonstrated the significant differences across different pre-school institutions bearing in mind that the day nurseries tend to take children from more deprived backgrounds. Anderson (1983), using the Pre-school Behaviour Questionnaire (PBQ; Behar & Stringfield, 1974), found a rate of 31.6%. The subjects were all children from economically disadvantaged homes which probably accounted for the higher rate.

Few studies have looked at the issue of pervasiveness of the problem across school and home situations. This may be because in the countries where studies have been carried out, a lot of the pre-school children (below five years) do not attend kindergartens. McQuire & Richman (1986*b*) used both a teacher's rating scale and a self-administered questionnaire for parents. They found that, as in the case of older children, the correlation between the scale and questionnaire results for pre-school children was not high, and only 3.8% were above cut-off in both instruments.

The validity of the questionnaire approach is open to the problems of rater bias, halo effect, social desirability effect, and reliability (Barkley, 1988). The semistructured interview approach probably has higher reliability and avoids the problem of perception bias of the rater. However, the significance of the symptoms cannot be evaluated – some children might be given a high score because they show a lot of minor problems. On the other hand, some children who are severely handicapped might score lower than the cut-off points because their dysfunctions are only shown in one or two areas of behaviour.

A clinical assessment by an experienced clinician is therefore important to validate the questionnaire or interview schedule and thus facilitate more accurate assessment of the prevalence of the disorder. So far, only Richman *et al* (1975) have used such a two-stage approach on a sample chosen randomly from the birth registry of a north London borough. They found 7.3% of the children showed moderate or severe behaviour disorder and 15% showed mild disorder.

The present study is a two-stage epidemiological study of behaviour disorder in Chinese pre-school children in Hong Kong. Wolf (1970) claimed that child-rearing practices are very resistant to change within the Chinese culture. Therefore even though Hong Kong is quite Westernised, the child-rearing practices may still be very different from those of Western countries. A cross-cultural comparison of the prevalence of behaviour disorder and the way these problems present is therefore of considerable interest.

Method

Screening stage

At the time of planning the study (September 1986), government statistics showed that 90% of the three-year-old children in Hong Kong were attending either kindergartens or child-care centres. All kindergartens in Hong Kong are run on a private basis and are under the supervision of the Education Department. The majority of child-care centres are administered by voluntary agencies or directly by government and are looked after by the Social Welfare Department.

The plan was to obtain a representative sample by first screening the kindergartens and child-care centres and for those not attending either of these types of institution (an estimated 10%), contact would be made through the registration of the Maternal and Child Health Clinics (MCHs). The limitation of this method was that those not attending either school or MCHs, a proportion estimated to be about 1% of the population, assuming that school and MCH attendance were independent, would not be covered.

Sampling based on MCH records alone was not feasible for two reasons. Firstly, although in Hong Kong over 90% of children under one year attend the MCHs, attendance drops sharply after the first year of life. Secondly, previous experience of trying to engage the parents and children from MCHs in a local language development study only achieved about a 50% success rate despite vigorous efforts to contact them (Ko, personal communication, 1986). Sampling on a door-to-door basis was also impracticable because the refusal rate was expected to be high.

Measures

The measures used in this stage included the PBCL, the BSQ and a semistructured interview on demography.

The PBCL is a 22-item checklist, designed specifically for use in group settings, which identifies children with behavioural or emotional disturbance. Each item is scored 0, 1, or 2 indicating that a particular behaviour is absent, present to a mild degree or present to a marked degree. There is a possible total score of 44, and a cut-off point of 12, identified as a result of observation, has been found to distinguish between children with and without problem behaviour (McQuire & Richman, 1986*a*). The questionnaire has been shown to be reliable and valid even when used by staff in different settings. The PBCL was translated into Chinese for local use. Forward and backward translations were carried out several times.

In a pilot study of two kindergartens and one child-care centre a total of 50 students were rated by five pairs of teachers to assess the inter-rater reliability of the PBCL. The product–moment correlation coefficient for the total score was 0.848. Two weeks later, five teachers rated the 50 subjects again and the test–retest reliability was 0.7.

The BSQ is a semistructured interview in which description of 12 types of behaviour are obtained from the parent. These include eating, bowel control, attention seeking and dependency, relationships with other children, activity, concentration, ease of control, tempers, mood, worries and fears. The validity and reliability of this instrument has been established (Richman & Graham, 1971). The behaviour items were scored on a three-point scale in which a score of 0 indicated an absence of the behaviour in question and scores of 1 and 2 were ordered categories of increasing severity of behaviour. The 12 items were summed to a total score with a range of 0–24. A cut-off point of 10+ has been shown to be appropriate for epidemiological studies (Richman *et al*, 1975).

To assess the reliability of the BSQ, after a period of training, two interviewers rated 20 subjects together, taking it in turn to do the interviewing. The inter-rater reliability was 0.907. They then separately reinterviewed another 20 subjects two weeks later. The test–retest reliability was 0.835.

The semistructured interview on demography covered parents' age, sex, occupation, income, education, place of birth, number of years in Hong Kong, and religion, the size of family, the ordinal position of the child, housing conditions, and the household income.

Procedure

The plan called for a sample size of 1000. Since it was calculated that 80% of three-year-olds were attending kindergartens and 10% child-care centres, the design was to sample 80 kindergartens and 10 child-care centres, stratified according to geographical region, whether private or aided, and the size of the school (small kindergartens to be clustered together), and to recruit 10 children from each. Letters were therefore sent to the kindergartens and child-care centres followed by telephone contacts. After obtaining consent, the school was asked to provide a list of the children whose chronological age was between 3 years 0 months and 3 years 12 months in that month. Random selections were then carried out to obtain 15 subjects in each school (10+5 reserves in case of parental refusal). The class teachers of the kindergartens or child-care centres were asked to fill in the teacher's questionnaire. Research assistants visited the school within days and interviewed the parents.

Starting from November 1986, about 10 kindergartens were screened every month. By June 1987, a total of 82 kindergartens had been screened (six of them were from clusters). In July 1987, 10 child-care centres and 1 special nursery were screened.

Attempts were made to reach the estimated 10% of children not attending kindergartens, nurseries or child-care centres through the MCHs. The plan was to contact a random sample of 1000 households with children of the right age via 10 randomly selected MCHs (stratified according to five geographical regions); from these 1000 children, it was expected that about 100 (10%) would not be attending either kindergartens or child-care centres and thus would complete the sample. In August 1987, therefore, a list of 300 households with children of the right age was prepared from each MCH since previous experience indicated a contact rate by telephone of around 33%. Telephone calls were made starting from the beginning of the list and moving to the next one if the telephone was engaged or unanswered, going back to the start when the 300 were exhausted. The telephoning stopped when 100 random contacts had been made for each MCH.

After an introduction, family members were asked on the phone whether their children were studying in a kindergarten or child-care centre or had been enrolled in a kindergarten or child-care centre for the coming September (the beginning of the academic year in Hong Kong). (Note that by August, usually all children will be enrolled for the coming year.) Only those doing neither of these were included in our study. The parents of these children were then interviewed in the MCHs following the same procedure as that for the kindergartens. Obviously, no information from a teacher was available.

Clinical diagnostic stage

First, a one in ten random sample was selected from the whole sample irrespective of their scores on the screening instruments. For the remaining children, all those who scored higher than the cut-off points in both the BSQ (10+) and PBCL (12+) were requested to join the second stage of the study. Also, a one in two random sampling was used for those children who scored higher than the cut-off point on either the BSQ or the PBCL. These cut-off points were used locally in our pilot studies and found to be acceptable.

Measures

The measures used in this stage included a parent interview on the symptoms of the child (PISC), interviews with the child, and the class teacher, psychological tests and various other measures.

The PISC is a semistructured interview based on the Parent's Account of Child Symptoms (PACS) developed in the Children's Department, Institute of Psychiatry, London (Taylor, 1986*a*). Modifications were made mainly to extend the schedule to include symptoms found in the pre-school years such as enuresis, encopresis and sleep problems. Also, the definitions of the frequency and severity of the symptoms were changed to cater for the fact that the PACS was being applied to younger children.

The interview with the child was a semistructured clinical interview modified from the interview schedule with children (Rutter & Graham, 1968). The child was first observed together with the mother. While the mother was being interviewed, the clinician provided the child with several toys to play with. These included a box of Lego, colourful cubes, a toy car, a puppet, and paper and crayons. Then, for a period of about 10 minutes, the clinician concentrated on talking to the child. He or she would assess the child's speech, activity level, fidgetiness, attention span, distractibility, disinhibition, mood problem, and relationship with examiner. The child would be asked to count, sort colour, draw a picture of a man, and given the names and functions of a few objects selected from a pack of name cards. A total of 24 ratings were made. At the end, an overall judgement of the psychiatric state was also made.

The interview with the class teacher covered four areas: 'learning', which included the performance of the child (i.e whether he or she appears to be slow in learning); 'behaviour', which included classroom behaviour such as any overactivity, inattention, disruption in class, non-compliance, withdrawn, mood problem; 'getting on with teachers', which included the relationship with the teacher, initiative in talking to the teacher and acceptance of the teacher's authority; and 'peer relations', which included acceptance by peers, co-operativeness and sharing, fighting, bullying or social withdrawal. A four-point rating (0–3) was made for each area (0 indicated an absence of problem; 1–3 were ordered categories of increasing severity of behaviour). The teacher's interview took 5–10 minutes to complete.

Other measures studied the birth and developmental history of the child, family and social factors, mental health of the family members, cognitive and language development of the child. They will be described in separate papers.

Psychological tests were used to examine the child's cognitive abilities and language development. Raters were blind to all the other information and the behaviour during the tests was recorded on a schedule modified from the child interview schedule. (Some of the items of the child interview were not applicable and they were excluded.)

Procedure

Within two weeks of the first stage, interviews with the parents and teachers, and clinical interviews with the children, were carried out by either a child psychiatrist or a clinical psychologist experienced in child psychology, blind to the results of the first stage of the study. In sum total, the interviews lasted for 75–90 minutes. The diagnosis was formulated according to predetermined diagnostic criteria, based on the symptoms only.

There have been very few studies concerning such clinical diagnostic criteria. Richman *et al* (1975) proposed criteria for assessment of behaviour disorder in pre-school children and provided a five-category subclassification along the dimension of severity (nil, dubious, mild, moderate and marked). However, the procedures on which these criteria were based did not include a psychiatric interview with the child. Controversy could occur if a clinician found no abnormality during interview but the parents or the school complained of symptoms in the child. The reverse situation could of course also occur although probably less often. Previous validity studies of a teacher's questionnaire using observation in school (McQuire & Richman, 1986*a*) or a parent's interview using home observation (Richman & Graham, 1971) showed that these procedures had good validity. Thus it seems unwise to ignore the information provided by the teacher's questionnaire or parent's interview, just because the child psychiatric interview does not reveal abnormality. On the other hand, clinical judgement should be based on objective data and not just the complaints of the parent or the teacher. Thus, for the present study, clinical judgements were based on the following:

(a) objective evidence, irrespective of parents' perception – detailed information of the severity and frequency of problem behaviour was obtained
(b) multiple sources of information – home, school and clinical interview
(c) Richman's original criteria – although impressions obtained in the clinical interview were not used to negate reports from the parents and teachers, abnormality in this interview was likely to increase the rating of the severity of the problem.

The reliability of the clinical diagnosis was tested in two ways. Firstly, for 10 cases, diagnoses based on the clinical case histories, which contained information from parents, teachers and the interview with the child, were made by five clinical professionals (three child psychiatrists, two experienced clinical psychologists) who were involved in this stage, and by Dr Naomi Richman who kindly agreed to participate in this part of the study. Diagnoses made by the five professionals were compared and the average weighted kappa (Fleiss, 1981) for these was 0.865 (s.e. 0.19). Dr Richman's diagnoses were compared with those of one of the authors (SL) and the weighted kappa here was 0.889 (s.e. 0.305). These results were very satisfactory. Secondly, during the actual field work, professionals went out in pairs from time to time to check their reliability, with one doing the usual diagnostic task and the other observing. The inter-rater reliability for a total of 14 pairs checked in this way was very satisfactory (average weighted kappa 0.859, s.e. 0.28).

Within two weeks of the interviews with the child psychiatrist or psychologist, a paediatrician and a clinical psychologist or speech therapist, who were blind to all the other information, visited the school to carry out tests on the language and cognitive abilities of the child (Reynell (1969) Language Test and Griffith's (1984) Scale D and E). These will be reported in a separate paper. The behaviour of the child during the tests was also recorded.

When all the information had been collected, the clinicians who had carried out the parent's interview contacted the parents and explained the findings to them. If the parents wanted help, a referral to a professional was offered. Most parents adopted a 'wait-and-see' attitude, but a total of four referrals were made. Also, all the parents were informed that the research team would contact them in about a year's time to follow-up the progress of the child.

Results

Screening stage

The final example consisted of 855 subjects selected from 82 kindergartens (of which six were from clusters), 10 child-care centres, 10 MCHs and one special school. We failed to obtain our targeted 1000 subjects by a fairly wide margin for two reasons. Firstly, out of the 1000 contacts from the MCHs, only five had children who were not enrolled at either a kindergarten or child-care centre. Of these five, two were autistic children, one was a recent arrival from China, one was looked after by grandparents in Macau (thus excluded), and only one was being kept back from schooling by parental choice. There are three possible reasons for this unexpected result, which reduced our sample by 96 subjects. Firstly, the 10% of children who did not attend schools may also not have attended MCHs (i.e. all those who attended school attended MCHs). A partial check at the second stage of how many children enrolled in school also attended MCHs gave a figure 90%, thus making this possibility unlikely. Secondly, the parents contacted through the MCH may not have answered truthfully. Although this is difficult to check, research workers noted no refusal, and all family members were co-operative on the phone. The third and most likely possibility is that the percentage of three-year-olds attending kindergartens and child-care centres was more than 90% and was approaching 100%. Thus our sample probably covered most of the three-year-olds in Hong Kong (except those not attending school or MCH, an estimated <1%) and with hindsight, the sample from the kindergartens should be slightly increased to make up for the difference (i.e. about 90 kindergartens instead of 80).

The second reason for not obtaining our target number of subjects was refusal by schools and parents to participate. In the case of the kindergartens, there were 38 refusals out of 121 approached (31%). For the child-care centres there was one refusal out of 11 approached (10%). For the MCHs, there were no refusals. The reasons for refusal were mainly that the school was too busy or that the headmaster felt it was not possible to obtain sufficient parental co-operation. To assess whether those schools which had refused were different in the rate of behaviour problems in the children, we requested these schools' teachers to fill in the PBCL for us. Out of the 38 schools, 21 (55%) agreed to this procedure. No significant difference was found in the overall PBCL scores of the children, suggesting no bias due to school refusals.

There were two kinds of refusal by parents. Firstly, some ($n = 43$, i.e. 5%) did not turn up for appointments (six from three nurseries and 37 from 26 kindergartens). Secondly, there were those who refused interviews. For each school or child-care centre, we had randomly selected 15 subjects – 10 and 5 for replacement if needed). For kindergartens, out of 804 subjects, 123 reserves were used. For child-care centres, out of 100 subjects, 15 reserves were used. The rate of using reserves was 138 out of 904, i.e. 15.3%. The effect of this refusal factor is that our sample was probably somewhat biased towards selecting mothers who were not working. Because of their work, some mothers probably found it harder to agree to interview.

Characteristics of the sample

The 855 children were seen by two interviewers, one seeing 548 cases, the other 307.

The interviewees included 734 mothers (85.9%), 59 fathers (6.9%), 40 grandparents (4.7%) and 22 other guardians (2.6%). The sex ratio of male:female subjects was 50.5%: 49.5%. The mean age of the children was 43.1 months (s.d. 2.9). Characteristics of their fathers were: mean age 35.5 years (s.d. 7.4); occupation – 34.2% in the semiskilled or unskilled categories; education – 61% below form 3 in secondary school; 41.6% were not born in Hong Kong and 13% had lived in Hong Kong for less than 10 years.

Characteristics of their mothers were: mean age 31.1 years (s.d. 4.2); previous occupation – 73.5% in the semiskilled or unskilled categories; education – 68.4% below form 3; and 40.4% were not born in Hong Kong. Of the families, 59.8% were either living in public housing or sharing a flat with others, 78.2% had a living area of less than 46.4 m^2, 71.3% had a household income of less than HK$7000, 25.8% had three or more children, and the mean number of children was 2.1.

When comparing the characteristics of the families of the study population with those of the general population few differences emerged. Firstly, only 30% of the mothers were employed compared with 53% of mothers aged 30 in the general population (1986 census). This difference is not entirely surprising, given that this is a sample of mothers with small children to look after and that 74% of them had unskilled or semiskilled jobs before, which can easily be returned to later. Also, our sample probably is slightly biased towards mothers who were not working. Secondly, our sample has fewer families with incomes below HK$2000 per month and also fewer in the higher income group (i.e >HK$10 000). The very low incomes in the general population are probably earned by single people or couples who cannot afford children while the high incomes are probably those who are past their child-bearing age.

BSQ and PBCL results

The mean value for the BSQ was 6.46 (s.d. 2.75) and 12.75% were above 10+. The mean for the PBCL was 8.78

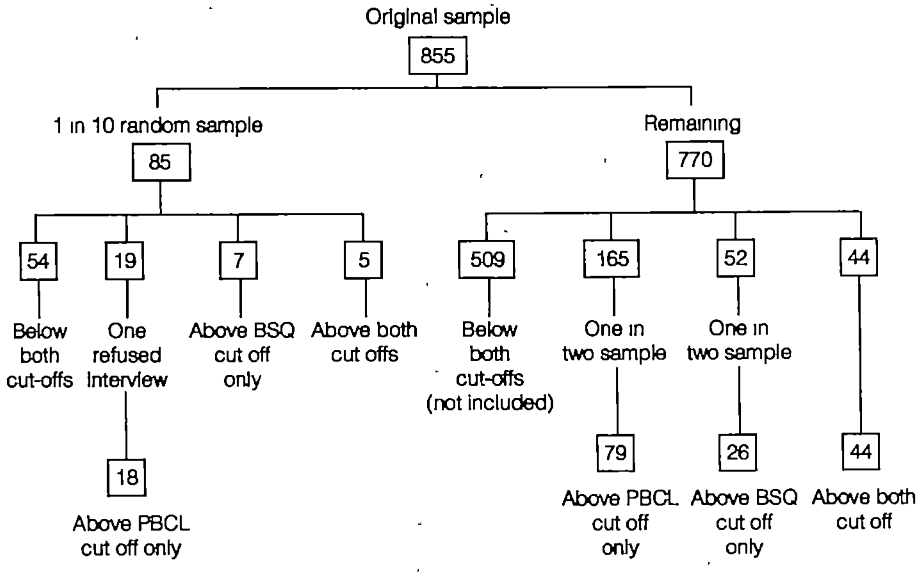

Fig. 1. Details of the sampling for the clinical diagnostic stage

Table 1
Breakdown of severity of behaviour disorder according to four groups formed by applying the cut-off points of the BSQ and PBCL

	Severity of disorder[1]					
	Nil	Dubious	Mild	Moderate	Severe	Total
No. below both cut-offs	38	12	4	-	-	54
No. above BSQ cut-off	7	7	16	1	2[2]	33
No. above PBCL cut-off	25	26	33	12	1	97
No. above both cut-offs	3	11	20	14	1	49

1. Richman's (1975) five-point category subclassification
2. Not attending kindergarten or child-care centre.

(s.d. 5.28) and 27.5% were above the cut-off of 12+. The product-moment correlation between the two instruments was 0.012, showing a poor correlation.

Combining the BSQ and the PBCL, 65.7% were below both cut-off points, 6.9% above the BSQ cut-off only, 21.5% above the PBCL cut-off only and 5.9% above both. These four groups served as the basis for further sampling for the second stage of the study.

Clinical diagnostic stage

Of the final sample of 234 subjects, one subject failed to turn up for assessment, leaving a total of 233. Fig. 1 shows details of the sampling.

A total of 85 subjects formed a random representative sample of the original 855 subjects. The distribution of children within this representative sample was very similar to that of the original sample. For the 233 subjects who formed the sample for the clinical diagnostic study, 49 were above cut-off points of both BSQ and PBCL, 33 were above BSQ only, 97 above PBCL only and 54 below both.

Prevalence of disorder in the study population

A breakdown of severity of disorder according to the four groups formed by applying the cut-off points of the BSQ and PBCL is shown in Table 1.

From these data, the prevalence of behaviour disorder in our population was calculated by combining the group proportions in the general population, estimated using the first stage sample, and the conditional rates within the groups estimated using the second-stage data. The result was: nil 53.6%, dubious 23.1%, mild 17.9%, moderate 4.55% and severe 0.75%. (For example the nil rate = (65.7% × 38/54) + (6.9% × 7/33) + (21.55% × 25/97) + (5.9% × 3/49) = 53.6%.)

Two cases of severe behaviour disorder were not attending school and therefore did not receive a teacher's rating. It would be reasonable to assume that they would also be picked up by a teacher if they were in a kindergarten or child-care centre. According to this reasoning, the prevalence was recalculated giving the following result: nil 53.7%, dubious 23.1%, mild 18.0%, moderate 4.5% and severe 0.7%, which made very little difference to the prevalence.

The distribution of severity of disorder according to sex is shown in Table 2. There was a very significant association of sex with behaviour disorder (Spearman correlation = 0.22, $P = 0.0009$), with more boys in the categories mild, moderate and severe.

There was no significant difference in the occurrence of diagnosis by geographical area (i.e. Hong Kong Island, Kowloon and New Territories). No statistical difference was found in the prevalence of behaviour disorders in kindergartens or child-care centres.

Validity of measures

The cut-off point on the BSQ of 10+, which appeared to be the optimal cut-off point, gave a sensitivity of 61.5% and specificity of 78.3%. The correlation with clinical diagnoses was significant (Spearman correlation = 0.38, $P = 0.0001$). With regard to the PBCL, the cut-off point of 12+ gave a sensitivity of 79.4% and specificity of 49.6%. The value of 12+ appeared to be the optimal cut-off point. The correlation with clinical diagnosis was significant (Spearman correlation = 0.35, $P = 0.0001$).

Results of psychological tests

As a partial way to validate the diagnosis, the behaviour of the child during the Reynell and Griffith's tests was examined. Behaviour was rated by testers blind to the clinical diagnosis, information about the family, and the child's behaviour at home and at school.

Table 2
Prevalence of behaviour disorder in Hong Kong pre-school children (36–48 months) by sex

Severity of disorder	Total	Male*	Female
Nil	54.7%	42.8%	63.8%
Dubious	22.1%	26.5%	20.1%
Mild	17.9%	23.5%	12.7%
Moderate	4.55%	5.5%	3.5%
Severe	0.75%	1.5%	0%

*Spearman correlation = 0.22, $P = 0.0009$.

Table 3
Comparison of children's behaviour in test situation across diagnostic groups

	Nil/dubious[a] ($n = 129$)	Mild[b] ($n = 73$)	Moderate/severe[c] ($n = 31$)	P value[1]	Pairwise comparison
Reynell language test situation					
Emotion/relationship factor: mean (s.d.)	1.33 (1.71)	1.85 (2.00)	2.84 (3.02)	0.0009	(c)>(a)**
Hyperactivity/non-compliance factor: mean (s.d.)	0.68 (1.29)	0.90 (1.40)	1.81 (2.50)	0.0005	(c)>(a)** (c)>(b)*
Griffith's test situation					
Emotion/relationship factor: mean (s.d.)	1.62 (1.63)	1.96 (1.79)	2.71 (3.02)	0.02	(c)>(a)**
Hyperactivity/non-compliance factor: mean (s.d.)	0.62 (1.26)	1.08 (1.88)	1.68 (2.17)	0.003	(c)>(a)** (b)>(a)*

*$P<0.05$, **$P<0.01$.

1 Analysis of variance.

Factor analysis of the various behaviour items in the test situation yielded two main factors. An emotion/relationship factor consisted of the following behaviours: addressing the tester, apprehension on entering the room, anxiety, emotional responsiveness, rapport with tester, smiling. A hyperactivity/non-compliance factor consisted of the following items: compliance, overactivity, fidgetiness, inattention, distractibility, disinhibition.

Comparison was made between the severity groups and the children's behaviour in the test situation as shown in Table 3. Significant differences were found between the moderate–severe group and nil–dubious group in all situations, whereas the ratings of the mild group were in between.

Discussion

Our results were fairly similar to those reported by Richman *et al* (1975). For the mild behaviour disorder, Richman found the prevalence in pre-school children to be 17–18% and for the moderate–severe disorder, 5.2–7.3%. As our sampling is not based on a birth-registry cohort, it is likely that we might have missed some children with moderate–severe problems who were not attending school or a MCH. Considering the likely difference in the child-rearing practices in the two cultures, this similarity of results is interesting. However, it may be a chance finding for which further replication of the results, especially in different cultures, is required before its implications can be considered.

The idea of pervasiveness has been emphasised in psychiatric disorders in older children (Schachar *et al*, 1981; Taylor *et al*, 1986*b*). It is also applicable in those of pre-school children. As most of our children were in a school after three years, we had the advantage of incorporating information from this into our assessment. Minde & Minde (1977) discussed the limitations of assessing pre-school children. Without the teacher's opinion, the assessment relies on the opinion of the parents who may have little opportunity to see their child in a wider social context. The young child often has not been exposed to group-based learning, making it not possible to assess the child's problem in full perspective. In our study the correlation of the teacher's questionnaire and parents' interview was very low. Similar results were found in other studies looking at teachers' and parents' ratings of the behaviour of pre-school children as well as that of older children (Langhorne *et al*, 1976; Minde & Minde, 1977; Goyette *et al*, 1978; Sandberg, 1981; McQuire & Richman, 1986*b*). This might reflect a situational effect, different styles of perception or different standards. The poor correlation made it imperative to combine the two instruments for the screening process. As far as we know, this approach has not been carried out before.

The prevalence of behaviour disorder according to the BSQ (cut-off point of 10+) was 12.75%, which was very similar to the results of other studies (Richman *et al*, 1975; Earls, 1980; Wolkind & Kruk, 1985; Corneley & Bromet, 1986). The prevalence of behaviour disorder as defined by the PBCL (cut-off point 12+) was higher than that found by McQuire & Richman (1986*a*). The specificity and sensitivity of the BSQ was much less satisfactory than the results reported by Richman & Graham (1971) of 91.2% and 70%, respectively. However, there was a major difference in method between the two studies since our study applied the BSQ to a community sample instead of a sample made up of clinical and normal cases. The former approach is known to be more demanding for a screening instrument. Later, Richman *et al* (1975) applied the BSQ to a community sample of 185 subjects and found a specificity and sensitivity of 90% and 92% respectively. In that study, the criteria for a case were mainly based on information from the BSQ, so it was not surprising that the BSQ had a high sensitivity and specificity. In our study, the diagnosis of a case was based on multiple sources of information and the clinician who made the diagnosis was blind to the BSQ scores.

The specificity and sensitivity of PBCL in this study also was not as good as that in McQuire and Richman's (1986*a*) original study. Using the same cut-off point, they selected 16 of the 24 'cases' and included 16 as 'cases' out of the 139 subjects of nil or dubious nature. Such a method gave a sensitivity of 66.7% and specificity of 88.5%. Admittedly, there are methodological differences between the two studies and the data are not strictly comparable. However, it is important to note that in our case the sensitivity was higher and specificity was lower. When we examined the PBCL score against clinical diagnosis, most of the false positives were found in the dubious group. It may be that teachers picked up differences in the children which were not sufficiently severe enough for a diagnosis of behaviour disorder.

The clinical diagnostic process for pre-school children has not so far been given special attention and does merit mentioning. In this study, following the usual clinical practice in child psychiatry, information was gathered from the child, the parents and the school. At the age of three to four years, the child is unlikely to be able to describe his emotions, relationships and the way he looks at his present state of development. One can, however, observe how he interacts with his caretaker, his play, his speech, his activity level and attention and his relationship with the interviewer in a particular social context. The contribution of the clinical interview of the child to the clinical diagnosis process will be dealt with in a separate paper.

In this study, the diagnosis of behaviour disorder was partially validated by its ability to predict abnormality of behaviour in the psychological tests. There was a significant sex difference in the occurrence of both the mild and the moderate–severe ratings. Male predominance in developmental disorders and hyperactivity is well recognised. However, for conduct and emotional problems in this age group, there are so far no epidemiological data suggesting a male dominance. Previous epidemiological studies which used the BSQ as the instrument to obtain information did not show a sex difference (Richman *et al*, 1975; Earls, 1980). The finding of no correlation between behaviour disorder and geographical regions was not surprising because Hong Kong is urbanising rapidly and there are now very few rural areas in Hong Kong. The finding of no difference between the kindergartens and child-care centres in the frequency of behaviour disorders was different from McQuire & Richman's (1986*b*) result. They had found a significant difference among play schools, nursery schools and day nurseries. In their study, the day nurseries admitted mainly children from disadvantaged families. In Hong Kong, child-care centres do not mainly admit children from disadvantaged families. Parents choose child-care centres mainly because they admit children at the age of two. Very often, by around the age of four, parents transfer their children to kindergartens.

Finally, in this paper, we subclassified behaviour disorder according to its severity. Whether the usual psychiatric subclassifications of conduct disorder, emotional disorder, hyperkinetic syndrome are applicable need further study. The established syndrome of autism and developmental disorder should be separated out. In our diagnosed cases of behaviour disorder, two who were not accepted by any kindergarten or child-care centre received definite diagnoses of autism. There were several other children showing autistic features. A further study of the subclassification of our subjects will be carried out.

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Sociocultural Correlates of Eating Disorders among Asian Schoolgirls in Bradford

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A survey of 204 south-Asian and 355 Caucasian schoolgirls was conducted in Bradford using the EAT-26 and the BSQ. At interview, seven Asian girls and two Caucasian girls met DSM–III–R criteria for bulimia nervosa, yielding a prevalence of 3.4% and 0.6% respectively. One Asian girl met DSM–III–R criteria for anorexia nervosa. Factor analyses of the EAT and BSQ supported their cross-cultural conceptual equivalence in this south-Asian population. Among the Asians, high EAT and BSQ scores were associated with a more traditional cultural orientation and not with greater Westernisation. It is probable that these findings reflect the cultural and familial difficulties faced by these Asian girls growing up in Britain.

It has been believed that anorexia nervosa and bulimia nervosa are uncommon in non-Western societies and among non-Caucasians in the Western world. There have been individual case reports of anorexia nervosa from Nigeria (Nwaefuna, 1981) and Zimbabwe (Buchan & Gregory, 1984). Postal surveys among psychiatrists in Malaysia and Japan have yielded rather more cases of anorexia nervosa. Buhrich (1981) reported 30 cases in Malaysia (19 Chinese, eight Indian, two Eurasian, one Malay). Suematsu *et al* (1986) found 1312 cases in Japan, a doubling of the prevalence in ten years. However, Lee *et al* (1989) report that anorexia nervosa is unexpectedly rare in Hong Kong.

A small number of cases of anorexia nervosa have been reported among blacks in North America (Warren & Van de Wiele, 1973; Jones *et al*, 1980; Hedbolm *et al*, 1981; Pumariega *et al*, 1984; Robinson & Andersen, 1985). In Britain, one Asian Caribbean and five Afro-Caribbean cases have been described (Kendell *et al*, 1973; Thomas & Szmukler, 1985; Holden & Robinson, 1988).

Bulimia nervosa has featured rather less in the psychiatric literature. In a survey of 50 Egyptian students in London and 50 matched students in Cairo, Nasser (1986) found six cases of bulimia nervosa (Russell's criteria) among the London sample but none in Cairo. Lacey & Dolan (1988) reported five cases of bulimia (DSM–III) among British blacks and Asians referred to their clinic in south London between 1982 and 1985. Holden & Robinson (1988) described 11 cases of bulimia nervosa among British blacks who presented at the Maudsley Hospital between 1979 and 1985.

Eating disorders are often linked aetiologically with the value placed on slimness in Western societies and its widespread pursuit by means of dieting (Nasser, 1988). Cross-cultural differences in the perception of female body shapes have been demonstrated by Furnham & Alibhai (1983) in a comparison of Kenyan Asians in Britain and in Kenya, and Caucasian British.

The Eating Attitudes Test (EAT; Garner & Garfinkel, 1979), has been used in several European countries to screen populations for eating disorders. Cultural differences in the EAT scores have been reported (Steinhausen, 1984, 1985). The only published report on the use of the EAT in non-Caucasians has been by Nasser (1986).

The Body Shape Questionnaire (BSQ; Cooper *et al*, 1987) has been developed as a measure of dissatisfaction with body shape, found in association with eating disorder. Items were derived from transcripts of interviews with anorexic and bulimic subjects. It has not previously been used with non-Caucasians.

Previous surveys of girls at school and in higher education in the UK have yielded a prevalence of anorexia nervosa of 0.2–0.4%, and of bulimia or bulimia nervosa of 1.5–2.0% (Crisp, 1976; Whitehouse & Button, 1988; Johnson-Sabine *et al*, 1988). No such survey has been conducted of girls from the ethnic minorities in the UK.

This study has three aims: (a) to assess the validity of the EAT and BSQ when used with Asian schoolgirls, (b) to estimate the one-year prevalence of anorexia nervosa and bulimia nervosa among Asian and Caucasian schoolgirls in Bradford, and (c) to identify the social and cultural factors which correlate with high scores on the questionnaires and with diagnosed eating disorders. We hypothesised: that there would be major discrepancies between Asian and Caucasian girls in their pattern of EAT and BSQ responses, giving these instruments limited cross-cultural validity; that the prevalence of eating disorders would be much lower among the Asian girls; and that eating disorders would only be found among the most 'Westernised' of the Asian girls.

In a previous paper we reported the prevalence of eating disorders among Asians and white schoolgirls in Bradford (Mumford & Whitehouse, 1988). This paper presents the factor analyses of the questionnaires and the social and cultural correlates of eating disorders in this population.

Method

This study was conducted in Bradford, an industrial city in northern England, with a south-Asian ethnic population of around 50 000 (1981 census). The subjects were fourth-year and fifth-year secondary-school pupils (aged 14–16 years) at four state schools in the Bradford metropolitan area, selected for their high proportion of Asian pupils. One school took only girls; the other three were mixed-sex schools. The proportion of ethnic-minority pupils in the schools ranged from 16% to 72%. This reflected both the ethnic composition of the particular metropolitan districts and also parental choice – many Muslim parents chose to send their daughters to the single-sex school, which had the highest proportion of Asian pupils.

A two-stage design was adopted for this study: a self-report questionnaire followed by selective interviews. The questionnaire consisted of three parts: personal and cultural data, the 26-item EAT (Garner *et al*, 1982), and the BSQ. These were administered in the classroom by teachers who had received instruction in their use. A glossary was provided for teachers to expand on the meaning of items in the EAT and BSQ that might cause difficulty; this had been developed during a previous pilot study with third-year pupils. Each girl weighed herself and had her height measured, and recorded these on the questionnaire form.

There was no available 'acculturation scale' appropriate to Asians in the UK. In an attempt to measure the 'Western' and 'traditional' cultural orientation of each Asian girl, four questions concerned with language, dress and food were devised for this survey and validated in the pilot study.

In the main study, subjects who scored above 20 on the EAT-26 or above 140 on the BSQ were invited to a structured interview using the Eating Disorders Examination (EDE; Cooper & Fairburn, 1987). This interview referred to the previous 12 months, and was conducted by two of the authors, who had received training in its use. Diagnosis of an eating disorder according to DSM–III–R criteria (American Psychiatric Association, 1987) was then made after detailed discussion between the authors. We have chosen to report the one-year prevalence since eating disorders fluctuate in severity, particularly the frequency of binge eating.

Results

Overall attendance in the classes surveyed was 69% of the number enrolled, as in the summer term some of the school-leavers were away gaining work experience. There was no reason to think that any of the girls were absent because of the survey. Eight girls of mixed or other ethnicity, or where ethnicity was in doubt, were not included in the study population. Thirty-seven girls (6.2% of girls in school) declined to take part. Six questionnaires were inadequately completed and also excluded. The remaining 204 Asian and 355 Caucasian girls constituted the survey population. Their age, weight and height, and body mass index (BMI) (weight divided by height squared) are given in Table 1.

Table 1
Age, weight, height, body mass index, dieting milieu and questionnaire scores

	Asians (n = 204)		Caucasians (n = 355)	
Mean age (s.d.): years	15.1	(1.6)	14 9	(1.0)
Mean weight (s.d.): kg**	48.7	(7 2)	54 1	(8.6)
Mean height (s.d.): cm**	157.9	(6.7)	163.0	(7 0)
Mean body mass index* (s d): kg/m^2	19 6	(2.9)	20.3	(3.0)
History of mother dieting in order to slim**				
never dieted	131	(64%)	146	(41%)
dieted 1–2 times	48	(24%)	102	(29%)
dieted 3 times or more	25	(12%)	107	(30%)
History of siblings dieting in order to slim				
no	145	(71%)	257	(72%)
yes	59	(29%)	98	(28%)
History of friends dieting in order to slim**				
no	92	(45%)	107	(30%)
yes	112	(55%)	248	(70%)
Mean EAT score (s.d.)**	10.6	(9.0)	7.7	(8.0)
Mean BSQ score (s.d)	73.3	(34.2)	70.3	(31 6)
No. scoring >20 on EAT (%)	25	(12.3)	31	(8 7)
No. scoring >140 on BSQ (%)	13	(6.4)	13	(3.7)

*$P<0$ 01, **$P<0.001$

Among the Asian girls in the survey, 179 (88%) were Muslim, 18 (9%) Sikh and 7 (3%) Hindu. The majority of Muslim families in Bradford have come from Mirpur District of Azad Kashmir, Pakistan. Of the Asian girls, 152 (75%) had been born in the UK. Of those born abroad, the mean age on arrival in the UK was 4.9 years.

Data were collected from each subject on any history of dieting by mother, siblings or friends. There were significant differences between Asians and Caucasians on two of these three variables (Table 1).

The EAT was scored using Garner & Garfinkel's (1979) system in which the three categories at the non-anorexic end of the six-point Likert scale are scored zero; the other categories score 1, 2 and 3 for each question. The BSQ is scored one to six on a Likert scale using the same response format as the EAT.

The mean EAT and BSQ scores of the Asian and the Caucasian girls are also given in Table 1, together with the number of subjects scoring over the predetermined thresholds on each questionnaire. There were no significant differences in mean EAT or BSQ scores between Asian girls

Table 2
Factor structure of the EAT-26

	Factor loadings Asians ($n=204$)	 Caucasians ($n=356$)
Factor 1, Dieting		
23 Engage in dieting behaviour	0.68	0.80
11 Preoccupied with desire to be thinner	0.66	0.71
1 Terrified about being overweight	0.65	0.70
7 Avoid foods with high carbohydrate content	0.63	0.55
12 Think about burning up calories	0.63	0.70
24 Like my stomach to be empty	0.61	0.40
16 Avoid foods with sugar in them	0.60	0.71
2 Avoid eating when hungry	0.58	0.55
6 Aware of calorie content of foods	0.56	0.73
17 Eat diet foods	0.52	0.77
14 Preoccupied with thought of fat	0.52	0.66
10 Feel extremely guilty after eating	0.49	0.62
22 Feel uncomfortable after eating sweets	0.45	0.72
Factor 2, Social pressure		
8 Feel others prefer if I ate more	0.70	0.78
13 Others think I am too thin	0.59	0.74
20 Others pressure me to eat	0.58	0.61
5 Cut my food into small pieces	0.54	
15 Take longer than others to eat meals	0.48	
Factor 3, Food preoccupation		
21 Give too much thought to food	0.70	0.64
3 Find myself preoccupied with food	0.69	0.70
18 Feel that food controls my life	0.58	0.60
4 Have gone on eating binges	0.47	0.65
25 Enjoy trying new rich foods	0.42	0.44
Factor 4, Vomiting		
9 Vomit after I have eaten	0.67	0.81
26 Impulse to vomit after meals	0.57	0.71
19 Display self-control around food	0.46	
24 Like my stomach to be empty		0.46

Total percentage of variance accounted for: Asians, 44.1%; Caucasians, 49.0%.
Missing values are factor loading <0.40

born in the UK and those born abroad, nor among the Asian religious groups (Muslim, Hindu and Sikh) or the Caucasians (Christian and Jewish).

Interviews were conducted with 22 Asian and 32 Caucasian girls, representing attendance rates of 73% for the Asian and 76% for the Caucasian girls invited for interview. Seven Asian girls and two Caucasian girls received a diagnosis of bulimia nervosa (DSM-III-R) (American Psychiatric Association, 1987), giving a one-year prevalence of 3.4% and 0.6% respectively: this difference was statistically significant (Fisher's exact test, $P<0.05$). One Asian girl and no Caucasian girls suffered from anorexia nervosa (DSM-III-R). Two Asian and four Caucasian girls received a diagnosis of eating disorder not otherwise specified (DSM-III-R).

Factor analyses of the EAT and BSQ

The method of factor analysis of the EAT used replicates that of a previous study of teenage schoolgirls (Wells *et al*, 1985). Inspection of eigenvalues in the light of Cattell's (1966) scree test suggested a four-factor solution for both ethnic groups. Oblique rotation was used. Table 2 shows those items loading above 0.40 on each factor for the Asian and Caucasian girls.

The responses to the BSQ were subjected to principal-components analyses which yielded four factors with an eigenvalue of more than one in both ethnic groups. Cattell's scree test did not offer a clear number of factors. Oblique rotation was used to determine a four-factor solution. Among both Asians and Caucasians, only factor 1 accounted for a substantial amount of sample variance in BSQ scores; it included most of the same items in both groups. The other three factors were different for each ethnic group and could not be interpreted.

To achieve a clearer delineation of the major constellation of BSQ items (factor 1), two-factor solutions were calculated. These yielded a slightly different set of items on factor 1 than the four-factor solution. However, when all the items are listed which achieved at least 0.40 loading on one or other solution, the list of items is identical for the two ethnic groups. We designated these items as comprising the 'principal common factor' (PCF) for the purposes of further data analysis. (Details of factor analyses available from the authors on request.)

In both ethnic groups, there was a substantial correlation between EAT scores and the principal common factor of the BSQ (Spearman's rho: Asians 0.57, Caucasians 0.51). The BSQ correlated with the BMI in both groups (Asians 0.39, Caucasians 0.40). The EAT correlated poorly with the BMI in the Caucasian girls (0.13, $P<0.05$); with the Asians, there was no significant correlation.

Table 3
Spearman's rank order correlations of scores for Western and traditional orientation (Asians only, $n=204$)

	Asian friends	Read Asian news-papers	Wear sari/ shalwa-kameez	English friends	English spoken at home
Do you read newspapers or magazines in an Asian language?	0.22				
Do you wear a sari or shalwa-kameez?	0.40	0.32			
Percentage of friends English	−0.43	−0.19	−0.35		
Is English spoken in your home?	NS	NS	−0.20	0.16	
Do you eat Western/English meals at home	−0.17	NS	−0.25	0.27	0.30

Critical value in two-tail test ($P<0.05$) is ±0.14

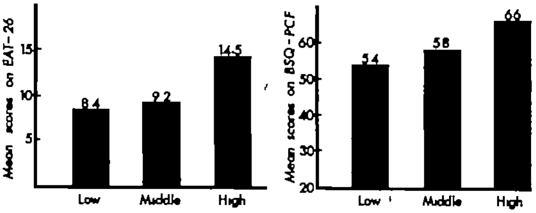

Fig. 1 Scores on the EAT-26 and BSQ principal common factor (PCF) for Asian schoolgirls scoring low, middle or high on two items rating cultural affiliation on a six-point Likert scale. The differences between groups on both the EAT and the BSQ are significant ($P<0.0001$, $P<0.05$, respectively).

Cultural correlations (Asians only)

The responses to the questions concerned with language, dress and food showed a consistent correlation pattern with each other and with the proportion of Asian and Caucasian friends (Table 3). The response to each item was given on a six-point Likert scale, identical to that used for the EAT and BSQ. The 'Western score' was defined as the sum of scores on the two Western items. The Asian girls were divided into three groups on the basis of their 'Western scores' and the mean scores on EAT and BSQ calculated for each group. No relationship was found between Western scores and either questionnaire.

The 'traditional' score was defined as the sum of scores on the two 'Asian' items. The Asian girls were divided into three groups on the basis of their 'traditional' scores. The most traditional Asian girls had the highest mean scores on both questionnaires, and the least traditional girls the lowest mean scores: these intergroup differences were statistically significant (Fig. 1).

A multiple linear-regression analysis was performed to test the hypothesis that traditionalism influenced eating pathology independently of the dissatisfaction with body shape. A stepwise multiple-regression equation was constructed with EAT scores as the dependent variable and BSQ, traditional scores and Western scores as the predictor variables. The hypothesis was confirmed. The regression analysis resulted in BSQ scores and traditional scores being entered as separate predictors of EAT scores; Western scores did not contribute significantly to the variance in EAT scores.

Discussion

All three instruments used in this study (EAT, BSQ, EDE) have been developed in the West for use with Western subjects. We were doubtful of their validity when used with these Asian schoolgirls in Bradford. We anticipated that Asian girls might place a different construction on certain items, or that other items, answered accurately, might have a different connotation and not be useful as indicators of eating pathology. For example, few Asian girls might rate strongly EAT item 8 ("Cut my food into small pieces") since knives are not commonly used when eating meals.

Determining the cross-cultural conceptual equivalence of questionnaires is a difficult issue (Lipsedge, 1989). Factor analysis has been recommended as a useful tool (Brislin *et al*, 1973; Irvine & Carroll, 1980). If one can demonstrate a similarity of factor structure of a questionnaire in two populations, this is strong evidence for cross-cultural conceptual equivalence.

The factor structure of the EAT in this study closely resembled that from a previous study of teenage schoolgirls (Wells *et al*, 1985). The dieting factor is identical across all three groups (Wells' sample, and Asians and Caucasians in this study). The other three factors show only minor differences between the groups.

There has been one previous factor analysis of the BSQ on a mixed population of 535 women in the community and 72 patients with bulimia nervosa (Taylor, 1987). This study also found one factor accounting for a substantial amount of sample variance. The items loading more than 0.40 on this factor were identical to the 'principal common factor' in Caucasians and Asians in our study, with one exception (item 28). The BSQ appears to measure a unitary dimension of concern with shape, best represented by our 'principal common factor'. No other factor has emerged in any of the three populations which is susceptible of meaningful interpretation.

The similarity of factor structures of the EAT and BSQ between Asians and Caucasians indicates that the comprehension of questionnaire items in both groups was similar. These findings are consistent with the EAT and BSQ having validity in this particular south-Asian ethnic group. It also suggests that these Asian girls have developed the same complex of eating attitudes and dieting behaviour (EAT) and dissatisfaction with their body shape (BSQ) as the Caucasian girls.

The choice of the 20/21 cut-off point for the EAT-26 in the Asian group is open to question. In the absence of separate validation data, the same threshold was used as recommended by Garner *et al* (1982) for Caucasian subjects. It should be emphasised that these Asian girls are not a 'Third World' sample. Most of them had been born in Britain; all of them had grown up in Britain and spoke English fluently.

Limitations on time prevented the interviewing of low scorers as well as high scorers. However, a recent survey of over 1000 girls in state schools in London (Johnson-Sabine *et al*, 1988) found that only 1% of girls scoring below the 20/21 threshold of the EAT-26 had eating disorders at interview, compared with 24% of high scorers. It remains a possibility that the optimal cut-off for either the EAT-26 or BSQ is lower for Asian than for Caucasian subjects, and that we failed to interview some Asian girls with eating disorders who scored below the threshold.

A further source of uncertainty both in questionnaire surveys and at interviews is the secretiveness and denial that are characteristic of anorexic and bulimic subjects. This is a confounding factor in all studies of eating disorders and is likely to operate in both Caucasian and Asian groups, perhaps to different degrees.

Prevalence of eating disorders

There have been two previous studies of the prevalence of bulimia nervosa in schoolgirls in the UK (Szmukler, 1983; Johnson-Sabine *et al*, 1988) and one study in a college population aged 16–19 years (Whitehouse & Button, 1988). Our finding of a one-year prevalence of bulimia nervosa (DSM–III–R) of 0.6% among the Caucasians is slightly lower than in these studies.

A prevalence study of anorexia nervosa in British schools yielded a prevalence of 0.2–0.4% (Crisp *et al*, 1976). No cases of anorexia nervosa (DSM–III–R) were found in this study among the 355 Caucasian girls. It would be misleading to extrapolate from the one Asian case of anorexia nervosa to any conclusion about its prevalence in that population.

Among the Asians, the very high prevalence of bulimia nervosa in this survey (3.4%) was unexpected. There has been only one previous case report of bulimia nervosa in an Asian girl in the UK (Lacey & Dolan, 1988). Our results in this particular south-Asian group (mostly Pakistani Muslims) suggest that eating disorders may be common among Asian girls in the UK. None of the bulimics had come to medical attention before our survey. Lacey & Dolan's finding of only a single case in a London eating-disorders clinic may reflect the poor uptake of elective psychiatric services by ethnic minorities in the UK.

It is not known how common eating disorders are across the Indian subcontinent, although their prevalence is probably very low. Private conversations with psychiatrists in several major cities of India and Pakistan indicate that sporadic cases of anorexia nervosa occur among Western-orientated social groups. Without adequate baseline data, we can only speculate that the high prevalence of bulimia nervosa among Asian girls in the UK is a new phenomenon in these families. It is probable that these Asian girls are increasingly adopting 'Western' patterns of reacting to personal conflicts and stressful life circumstances.

There is a parallel to our findings from studies of the parasuicide rate among ethnic minorities in Britain. A recent study in Birmingham found a greater rate of self-poisoning among young Asian-born women compared with all British-born women (Merrill & Owens, 1986). A previous study in Birmingham found that the rates among Asians were lower than among Caucasians (Burke, 1976). This increase in the rate self-poisoning suggests both an increasing adoption of Western patterns of reacting to stress and also high levels of distress in these young Asian women.

The Asian girls' higher EAT scores indicate that they had greater concerns about food intake and weight than the Caucasian girls. Such concerns, and the high prevalence of bulimia nervosa in these Asian girls, cannot be explained in terms of excessive concern with body shape, since their scores on the BSQ were not significantly different from those of the Caucasian girls. This seems to contradict common stereotypes about Asian attitudes to female body shape and suggests that the higher prevalence of eating disorders among the Asian girls has to do with factors other than their dissatisfaction with body shape.

Social and cultural factors

The Asian girls who make greatest use of Asian language and dress had the highest mean scores on both the EAT and BSQ. The use of English in the home and consumption of Western food showed no relationship with questionnaire scores. Moreover, those Asian girls diagnosed as bulimic had significantly higher 'traditional' scores than the rest of the Asian girls; their 'Western' scores were not significantly different.

Various explanations could be advanced to account for these surprising results. The most traditional girls may be experiencing the greatest internal conflict, for example around issues of identity as they grow up with two sets of cultural values. It might be that the greater the difference between the two cultures, the greater the internal conflicts and anxieties which arise. Another contributing factor could be greater rigidity of family functioning within the more traditional families, leading to greater intergenerational conflict (widely held to be an aetiological factor in Western anorexics).

The items used in this study to measure cultural orientation were chosen as being simple and factual. The questions have face validity, concurrent validity with the friendship network, and showed a consistent correlation pattern with each other. However, the items should be regarded as no more than indicators of a complex cultural phenomenon. There is a need to develop a more sophisticated measure of these cultural dimensions. It may be of significance that the 'traditional' and 'western' scores showed only a modest inverse correlation (–0.2), suggesting that traditional and Western orientations do not lie at either end of a single continuum. Some families might incorporate both sets of attitudes and values strongly. It is probably better to visualise traditionality and Westernisation as occupying orthogonal axes, analogous to the model of 'four modes of acculturation' proposed by Berry (1988).

A study of acculturation and eating attitudes among Hispanic adolescent girls in the USA has been reported by Pumariega (1986). His 'acculturation questionnaire' contained similar items to our study, although he assumed that 'English' and 'Spanish/Latin' orientations were located on a single continuum. This study found a significant positive correlation (0.18) between EAT scores and acculturation, lending support to his hypothesis that greater adherence to the prevalent US culture may increase an individual's vulnerability to developing eating disorders.

The closest parallel to our findings comes in two studies from Kuwait on the relationship between intergenerational conflict and psychiatric symptoms (El-Islam *et al*, 1986, 1988). Modern Kuwait had a number of similarities with the Mirpuri Muslim community in Bradford: a conservative Muslim society experiencing rapid social change by reason of exposure to Western attitudes and values. El-Islam *et al* found a tendency towards a greater number of psychiatric symptoms (as measured by the 28-item General Health Questionnaire in Arabic) in association with greater *difference* in cultural attitudes between parents and children. There was no such relationship between symptoms and 'liberal' attitudes. He suggests that it is conflict of attitudes, rather than the adoption of non-traditional attitudes as such, that is likely to be associated with psychiatric symptoms.

From his clinical experience, El-Islam (1983) states that the intergenerational conflicts centre mainly around patterns of family relationship, methods of marriage and the emancipation of women (El-Islam, 1983). Various explanations for these conflicts have been offered by Arab commentators, summarised by El-Islam: parental attempts to direct children in accordance with parents' aspirations rather than their own interests, exposure to Western influences, adoption of more progressive values by the young, rigidity of parental control or loosening of parental control.

These themes figure prominently among Muslim young women who are referred to the psychiatric services in Bradford with neurotic disorders. They are well illustrated in an account written by a 19-year-old girl from a Pakistani Muslim family in Bradford who developed anorexia nervosa (Appendix). It is probable that the increased prevalence of eating disorders among these Asian schoolgirls is just one indicator of hidden conflicts and distress experienced by many Asian girls from traditional families growing up in Britain.

Appendix

"I am facing what is called 'culture clash'. Because I have been educated in England I have been taught to be independent, original, to think for myself and to be successful in my life by achieving what I aim for. In school I was prepared how to handle my life and how to stand up for myself, while when I went home I was expected to be quiet, submissive, obedient and totally dependent on my parents.

"Like most Asian parents, my parents believe that only they know what is best for me. That is why they find it hard to cope with me not being happy with their decision about my marriage. My parents see the breakdown of my marriage as totally my fault. They believe girls should not speak up and should let their parents decide what happens in their life. Asian culture has a lot of influence over how older people think and it is because of the culture that I am expected to sacrifice my life and happiness for the sake of my parents and family's honour and respect.

"My parents treat me as if I have been a big failure to them – I am seen as unworthy and unreliable and get treated as if I am a rebel. It is hard enough for me to make a decision on what I am going to do in the future without the added burden of thinking of the family and their images. I feel trapped most of the time anyway, because no matter what I do I will end up losing in one way or another. I know I am caught in the 'Catch-22' situation. If I do as my parents ask I will lose out on my own life and if I do as I please I would probably lose my family. I do not know why I should do as the parents and family ask – they have all enjoyed their lives and now they want to control my life.

"There are times when I fell really guilty about causing all this trouble. I feel weighed down and depressed most of the time and the only way I can pick myself up is by thinking of how I would like things to be or how far I would have got in five years' time. . . . I will never give in to them though: there is no way I can live under their thumb. I do not know where I stand with my family and relatives; I feel confused when my relatives are around, but I have still got some fight left in me yet."

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Anorexia Nervosa in a Group of Asian Children Living in Britain

RACHEL BRYANT-WAUGH and BRYAN LASK

Four cases of anorexia nervosa occurring in Asian children are described. These case histories are set against the recent increase in eating disorders in patients of different racial origin. The role of sociocultural conflict in immigrant Asian families to Western countries is raised as a possible contributor to the emergence of eating disorders and the need to be aware of anorexia nervosa in such childhood populations is stressed.

It has been claimed that anorexia nervosa is virtually limited to patients of white racial origin (Russell, 1985). Russell believes that this observation cannot be dismissed as being due to limited levels of medical services or poor statistical returns in developing countries, because cases are seldom reported among non-white immigrants in Western populations. Halmi *et al* (1977) stated that "there is not a single well documented case in the literature of this illness occurring in a black". Crisp *et al* (1976) found no black subjects in a survey of nine British schoolgirl populations, while Hall (1978) found none in a similar study carried out in Australia.

In contrast to the findings of these studies dating from the 1970s, reports from the 1980s suggest that anorexia nervosa may be becoming more common in patients of various ethnic origins (Hsu, 1987). It is possible that this apparent increase in the incidence of anorexia nervosa in both immigrants to Western countries and non-white patients in their country of origin, may be related to a general increase in the incidence of anorexia nervosa. Anorexia nervosa has been well documented in Japan (Suematsu, 1985), in blacks in the United States (Pumariega *et al*, 1984; Silber, 1984; Robinson & Andersen, 1985; Hsu, 1987), Britain (Holden & Robinson, 1988) and Africa (Nwaefuna, 1981; Buchan & Gregory, 1984), and in Asians in Malaysia (Buhrich, 1981) and Singapore (Ong *et al*, 1982). It has also been reported in adolescent Vietnamese refugees (Kope & Sack, 1987), in Arab students in both Britain and Egypt (Nasser, 1986), in two Arab children (Lask & Bryant-Waugh, in preparation), and in patients of Afro-Caribbean origin (Thomas & Szmukler, 1985). Normal-weight bulimia has also been reported in British and American blacks and Asians (Lawlor & Rand, 1985; Lacey & Dolan, 1988), and in Asian schoolgirls in Britain (Mumford & Whitehouse, 1988). Hsu (1987) concludes that there is no longer any doubt that typical anorexia nervosa does occur among blacks.

In our original studies of childhood-onset anorexia nervosa (Fosson *et al*, 1987; Bryant-Waugh *et al*, 1988), we did not identify any Asian children. However, over the subsequent three years, 4 (13%) of 30 successive referrals of girls with anorexia nervosa were of Asian origin. All fulfilled both DSM-III (American Psychiatric Association, 1980) and ICD-9 (Rutter *et al*, 1975) criteria for anorexia nervosa.

Case reports

Case 1

MI was a 13-year-old girl born in Bangladesh, the elder of two children who came with their parents to live in England during their early childhood. Both parents came from poor Bangladeshi families, but had achieved relatively high standards of education. M's father had first visited England in the 1960s to further his studies. He had remained for seven years before returning to Bangladesh for an arranged marriage to M's mother. Shortly after the birth of their second child the family emigrated to England. Mrs I had not previously travelled outside Bangladesh. Both parents felt they have had difficulty settling, and both still experience language problems. Mr I was in hospital for a month suffering from depression shortly after immigration.

M and her younger sister both spoke good English, and attended local English schools. M often became impatient and embarrassed regarding the parents' poor command of English, especially that of her mother. Mr I was very involved with a local cultural centre for Bangladeshi families and insisted that the rest of the family participate in the centre's activities. M and her sister often attended against their wishes, although their mother was happy to do so. Mrs I socialised primarily with other Asian families and relatives who lived nearby, and had very little contact with English neighbours. The children had a mixture of English and Asian friends, although tended to keep the two separate, and did not like to bring their English friends home.

M was born after a planned pregnancy. She was reported to have been a poor feeder from birth, and in infancy required nasogastric feeding for several weeks. All other aspects of her development were reported to be normal. At the age of eight she developed cold hands and feet, and eventually she was diagnosed as suffering from Raynaud's disease. In the year before referral she became very much more fussy about eating, refusing to take meals with the family and expressing a fear of becoming fat. At school, a number of her friends had been on diets. Her mother had tried to encourage her to eat by preparing elaborate Indian dishes and insisting that she should eat certain amounts of rice each day. M refused, claiming that Indian food was very fattening. Menstruation, which had started at the age of 11, ceased shortly before her 13th birthday. She was described as a highly conscientious pupil at school and had an IQ of 140. She displayed a number of obsessional features at home, centred around homework, personal hygiene and cleanliness.

On examination, MI was dehydrated and emaciated, her weight for height being 76% of normal using the Tanner–Whitehouse standards (Tanner *et al*, 1966), where 80% or less indicates wasting (Cole, 1979). She had lanugo hair, poor peripheral pulses and a blood pressure of 90/65 mmHg. Secondary sexual characteristics were present, but with minimal breast development. She was depressed, had a distorted body image, and expressed a fear of getting fat. She refused to eat or drink. An ultrasound scan of her ovaries showed regression to infantile size and configuration.

Treatment consisted of firm encouragement to eat, with gradual introduction of increasing quantities of food and fluids, reward schemes for weight gain, parental counselling, family therapy, individual psychotherapy and the use of the ward milieu to help her better understand the origins of her illness and to find ways of coping better with contributory stresses, such as intense family pressure. Cross-cultural conflicts were addressed in family therapy sessions and formed an important focus for intervention.

MI made a slow recovery and was discharged six months after admission, having regained normal weight and behaviour. Unfortunately, she relapsed within a few months and required a second admission of seven months. At six months after the second discharge, she had maintained her weight, but her periods had not recommenced. She was, however, coping very well at school and in her peer group. In addition, both M and her parents appeared to be more tolerant of previously conflicting cultural issues.

Case 2

DH was a 13-year-old girl born in England to Indian parents. Mr H was reluctant to enter his arranged marriage because his prospective wife was illiterate. He arrived in England in 1963 and his wife joined him four years later. Mr H attended evening classes and obtained qualifications to become a plumber and heating engineer. Mr and Mrs H had a poor marital relationship, with Mr H often openly expressing contempt for his wife. Mrs H spoke very little English and did not know many people outside the family. She did not like living in England and would have preferred to return to Bangladesh.

Mr and Mrs H had two children besides D, both older boys. The oldest, R, 18 months before D's referral, married an Indian girl brought up in England; but continued to live, with his wife, as part of the H family. This was not an arranged marriage and was against his parents' wishes. D's new wife was not much liked by other members of the family, and was described as a 'trouble-maker'. Since the wedding there had been frequent stormy arguments between R and his parents. The younger brother, P, aged 16, was still at school. The two younger children were born in England; all three had received an English education, and spoke much better English than their parents.

One year before D's referral the family acquired a small general store where they all worked. It had been a difficult year, and Mr H had felt under considerable strain. D and her younger brother P were expected to help with the business after school and at weekends. Consequently, they had little leisure time, and were aware that their school peers led a very different lifestyle. Although D usually co-operated, P had often refused to work in the shop, and had taken to staying out without informing his parents of his whereabouts.

DH was born following a planned pregnancy and forceps delivery. There were no particular early problems. Feeding and general development were reported as normal. About three years before referral D was noted to become increasingly shy and timid, both at school and with people outside the family. She was reported to be eating very little at home, and was not seen to eat at school. She continued to do well at school, but the problems with shyness and eating appeared to worsen after her transfer to secondary school. Eventually she stopped attending school as she was unable to speak to anyone there, and Mr H taught her at home. By the time of referral she was reported to be eating very little, and only when encouraged by her family. She was also reported by family members to engage occasionally in self-induced vomiting.

On examination D was uncooperative and extremely shy and embarrassed. Her Tanner–Whitehouse weight for height was 76%. She had lanugo hair on her back, poor peripheral pulses and a blood pressure (sitting) of 100/70 mmHg. Secondary sexual characteristics were present, and menstruation had been absent for the previous seven months. She considered herself to be overweight and expressed a fear of becoming fat. An ultrasound scan revealed an adult-sized uterus but some regression of the ovaries.

Treatment consisted of admission to hospital to restore normal weight and eating patterns and to work on family issues and social skills. A behavioural programme was introduced involving rewards contingent on weight gain. It became clear that D was extremely concerned about her future. She perceived both her mother and her sister-in-law to be unhappy and looked down upon by the male members of her family. She associated this with the fact that they were of Indian origin, and in turn felt that she was relatively worthless. She worried that her future existence would be a burden to others and herself.

Unfortunately Mr H withdrew D from the ward after only two weeks of treatment. During this time she had not gained weight, and appeared to be outwitting staff by vomiting in secret. Mr H refused to allow his family to attend for family-therapy sessions and considered the therapy offered to be generally unhelpful. The H family's attendance had been erratic, with only a few members being present at any one session. Clearly the running of the family business was a major priority, and attempts were made to come to mutually acceptable arrangements. These attempts failed and the H family did not reattend.

A letter was received from the referring physician some months later, stating that D remained seriously ill. The family was again offered an appointment but failed to respond.

Case 3

BL was a 14-year-old girl referred by her school doctor with a suspected diagnosis of anorexia nervosa. She attended a girl's school which placed a strong emphasis on academic success. The school doctor reported that B had a distorted body image, experienced guilt about eating, and engaged in purgation and self-induced vomiting. Weight loss over the nine months before referral had been about 13 kg. Although, according to the school doctor, B's parents were aware of the extent of the weight loss, B had insisted that there should be no contact between the school and home. In addition, B had refused to see her general practitioner regarding the problem. By the time of referral, however, she had begun to accept that she needed some help.

On examination, a diagnosis of anorexia nervosa was confirmed. In addition to the features mentioned by the school doctor, B had a very restricted diet, a fear of becoming fat, and secondary amenorrhoea. She was also found to be engaging in excessive exercise, primarily at home. At school she was a keen sportswoman. B was a very pleasant, articulate girl who was clearly very anxious to please her parents and stay on good terms with them. Food was reported to be the only area of conflict within the family. During the assessment interview, both B and her mother became very upset. Both parents stated that they would find it extremely difficult to be firm with their daughter at mealtimes, and both claimed that they considered her generally 'alright'.

Both of B's parents were born and brought up in India. They had an arranged marriage and had moved to England in their 20s in order to improve their standard of living. B was a baby at this time, having been born in India. The parents had no further children and reported that they felt they had settled well in their new environment. At the time of referral, Mr L worked as a laboratory assistant and Mrs L did office administrative work. Both parents spoke good English and had a mixture of both English and Indian friends. They had no close relatives in England and were frequently in touch with family in India. The family observed Hindu religious practices, and B was particularly eager to participate in the rituals of the religion.

In the summer before referral the L's had travelled together to visit relatives in India. This was B's first visit, which she said she had much enjoyed. It became clear that the extent of the poverty she had witnessed and the large numbers of very thin people she had seen had made a profound impression on her. She said that she had felt fat at this time, and that various members of her extended family had made remarks about her appearance.

On her return to school after the summer, a friend had teased her for being fat. This friend had also wanted to talk about boyfriends, which made B feel uncomfortable. The friend's remarks together with B's experience in India appear to have triggered the onset of dieting. B stated that her aim in life was to become a surgeon and then to live in the United States. She added that she would visit India once a year.

B was treated on an out-patient basis using a combination of family therapy and individual work. Issues covered were B's sense that she had to be perfect, her unease about her own sexual development, and her gradual separation from her parents. She was helped to make sense of her experience in India, and to integrate her 'Indian self' with her 'English self' (she felt that because she was Indian she should also be thin and poor, and in some way had to make up for the fact that she was not). Responsibility for weight gain was placed, after negotiation with the family, largely in B's hands, with the parents providing monitoring when necessary.

B responded extremely well to this programme. She gained weight slowly but steadily until her target weight had been reached. She made good use of therapy sessions, and appeared to be resolving her difficulties with her own cultural identity. By the time of discharge, menses had resumed, and both parents reported that she was eating sensibly. Mr and Mrs L remained quite overprotective of their daughter, but were aware of this and worked hard to keep this in check. B was in addition engaging more with her peer group and appeared more relaxed in male company.

Case 4

PS was a 13-year-old girl referred to the psychiatric department by one of the hospital's paediatricians. She had been admitted to a paediatric ward for investigation of weight loss and vomiting, in the context of a family history of tuberculosis. No organic cause could be found and a diagnosis of anorexia nervosa was suspected and later confirmed.

P had become very conscious about her weight and figure after staying with her uncle's family in the summer before referral. She felt that her clothes were becoming too tight, and decided to diet. She complained that she felt full after eating and frequently vomited. Her parents took her to a non-medical practitioner who massaged her abdomen and provided some explanation for her symptoms. Six to eight weeks after this P stopped overt vomiting, but was thought to have continued vomiting in secret.

By the time of referral to the paediatric ward, P was emaciated and depressed. During her admission she gained a small amount of weight, and when seen in the psychiatric department was under 80% on the Tanner–Whitehouse weight for height. She appeared very uneasy talking about her attitudes to growing up and puberty.

P was the eldest of five children born to Indian parents. P and her sister were born in India before the family emigrated to England, where the remaining three children had been born. Mrs S spoke virtually no English. Mr S spoke and understood more, but the family required an interpreter. Owing to the difficulty in obtaining the services of a hospital interpreter, the S's eldest nephew fulfilled this role.

Mr S was a bus driver and Mrs S was a part-time cleaner. P as the eldest daughter spent a lot of time engaged in domestic chores and looking after her siblings. She was expected to fulfil these duties before going out or meeting friends. Mrs S and P had frequent arguments about clothes, and P's parents determined to a large extent what she was and was not allowed to wear. Mrs S always wore a sari, and considered many Western clothes immodest. Mrs S was also a keen cook; she spent many hours preparing the family meals and expected P to do the same. P resented the time she had to spend on household chores, and felt it unreasonable of her parents to be so restrictive regarding her dress and leisure activities.

The S family were seen on three occasions in the psychiatric department, and admission was strongly recommended. Mr and Mrs S originally agreed to this, although P was very much against it. She became angry with her father and accused him of wanting to get rid of her. On the day of the planned admission the S family failed to attend. P exerted a very powerful influence on her parents, resulting in her father becoming exasperated and her mother tearful. Unfortunately, despite repeated efforts, the family did not reattend. P remained resistant to the idea of returning to the hospital. It proved impossible to obtain any follow-up information.

Discussion

These cases of undisputed anorexia nervosa in four Asian children (mean age of onset 12.6 years) do not support the view that this illness is restricted to white patients, and add to reports of anorexia nervosa in other racial groups. Further, to our knowledge they are the first reported cases of anorexia nervosa in Asian children. Since preparing this paper we have seen four more such children with anorexia nervosa.

We agree with the suggestion that culturally determined attitudes may interact with psychological variables in the genesis of anorexia nervosa (Garner & Garfinkel, 1978). In societies where food is plentiful, the ideal of slimness is constantly imposed via the media, advertising and peer-group pressure. In developing countries where food may be scarce, the ideal of feminine attractiveness is often the opposite to slimness (Furnham & Alibhai, 1983). The children of immigrants from such countries, although sharing their parents' and grandparents' physical and psychological characteristics and heritage, may be exposed to a different and conflicting set of sociocultural norms and ideals.

In case 1, cultural conflicts were clearly present in the form of the father's insistence that the children participate in the activities of the local Bangladeshi cultural centre, whereas they preferred to spend their free time with school friends, engaged in pursuits not considered part of a traditional Asian upbringing. In case 2, cultural conflicts were present in the form of the children's superior knowledge of English, D's distress at the prospect of adult life modelled upon her mother's compared with that of the mothers of her school peers (and her fear that she had no way to escape this), and the clash of values carried from home to those observed at school. In case 3, there was difficulty in integrating Asian heritage and family with an English lifestyle, and in case 4, domestic expectations, use of leisure time and choice of dress formed important areas of conflict. The more 'traditional' the family, arguably the greater is the possibility of sociocultural conflict around such issues as arranged marriages, norms regarding dress and contact with the opposite sex, the role of females, and mealtimes and cooking. It is of interest that we have not yet seen an Asian child with anorexia nervosa from a family who seem to have exchanged their traditional culture for a more typical Western lifestyle.

Anorexia nervosa has frequently been associated with difficulties imposed by the developmental demands of adolescence. Crisp's (1980) view of anorexia nervosa as essentially a psychological disorder resulting from an avoidance of adolescence is a well known example of such a developmental theory. The common tasks of adolescence, which include the formation of an integrated sense of self, the emergence of an independent self, and the acceptance of a sexual self, are undoubtedly problematic for many young people. It can be argued that issues of autonomy, control and sexuality may present particular difficulties for children and adolescents such as those described in this paper. Given that norms and social rules regarding such issues are largely culturally determined, it is not difficult to envisage that young people growing up in a situation involving a juxtaposition of two very different cultures may experience confusion. This in turn may make them more susceptible to the development of eating disorders, a process which has often been linked to confusion regarding the individual's sense of self (e.g. Bruch, 1973). DiNicola (1990) has proposed the transcultural hypothesis that anorexia nervosa can be viewed as a 'culture change syndrome' whose onset may be triggered under conditions of 'sociocultural flux'. He cites case examples reported by Bulik (1987) and Kope & Sack (1987) which support this notion, and illustrate that stresses related to immigration and acculturation may lead to the emergence of eating disorders in populations not previously considered at risk.

We suggest that the alternative influence to which these children are exposed, with resulting intercultural and intrafamilial conflicts, may act as additional stress factors contributing to the development of anorexia nervosa. We need to be aware of the existence of anorexia nervosa in young people from families of different racial backgrounds, and of the possibility of an increasing incidence, for delay in diagnosis is far too common, with the danger of subsequent poor prognosis (Fosson *et al*, 1987).

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Child Physical Abuse Observed
Comparison of Families With and Without History of Child Abuse Treated in an In-patient Family Unit

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Twenty-seven families, 14 with a history of child physical abuse and 13 with no such history, were studied over the course of intensive in-patient treatment. The families in the former group differed significantly from those in the latter group in terms of current circumstances and background histories. Families where abuse was admitted benefited significantly more from treatment than families where abuse was suspected but not admitted. The ability of mothers to remember good relationships from childhood and to establish good relationships during treatment was an important prognostic factor for successful treatment.

The Cassel Hospital, part of the National Health Service since 1948, provides in-patient psychotherapy for disturbed adults, adolescents and families (Denford, 1986; Kennedy *et al*, 1987). The Families Unit at the Cassel Hospital is a unique in-patient treatment resource that facilitates observation and direct experiencing by staff of disturbances in family life and functioning. An average of 20 families are assessed and treated as in-patients every year. Increasingly, disturbed and disturbing families, where abuse of a child has occurred, are referred for treatment (Kennedy, 1988).

As an area of scientific investigation, child abuse is still a riddle, despite a great investment of energy and funds over the past 20 years. The literature on child abuse and neglect is enormous. However, integrating the findings of diagnostic evaluation into a treatment programme is perhaps the most difficult step in working with the abused child and his or her family. There are relatively few studies to guide us in choosing interventions which best help the family to change (Leventhal & Midelfort, 1986). Cohn (1979), in a review of 11 treatment projects, found that only about 40% of over 1700 patients were felt to have shown a decreased potential for abuse, despite multiple interventions by several professional and lay support staff.

The Cassel Hospital Families Unit is a setting in which the diagnostic evaluation of family functioning is closely linked with the treatment programme (Kennedy *et al*, 1987). The safety of the child is central in thinking with the family about the work of the day, about relating within the family, and about interactions with others. The strengths and pathology of families, and of individuals within families, are actively experienced by therapist, nurse and others. Understanding this phenomenon of transference, and the corresponding countertransference reactions, adds a further dimension to observing these families who live, love, work, fight, hate and survive within the holding structures of a psychoanalytically orientated hospital.

The study reported here is a naturalistic and descriptive clinical investigation, similar to a previous study of in-patient psychotherapy at the Cassel Hospital (Denford *et al*, 1983). Aspects of the lives of families admitted for assessment and treatment of various disturbances in family functioning are explored. Families where child physical abuse is definite or suspected are compared on a number of dimensions to families where child physical abuse is not suspected. Specifically, the histories of parents, the quality of their lifetime relationships, and the changes made by the family during treatment are contrasted. The 'null hypothesis', that there is no difference between abusing families and non-abusing families as experienced in this setting, is tested where appropriate.

Method

All 27 families admitted to the Cassel Hospital for assessment and treatment during the 18 months from May 1986 to October 1987 inclusive, were studied. There were nine two-parent families and 18 single-parent families. Family size varied: there was no child in one family, one child in 15 families, two children in six families, three children in three families and four children in two families.

An abusing family was defined as a family with one or more child in care and/or referred by social services, and one in which concern was expressed by the referrer and/or Cassel worker about the safety of a child. In seven of the 14 families in this child abuse (CA) group, a child (four girls and three boys) had definitely been physically abused (definite abuse (DA) group). The injuries inflicted consisted of bruising and soft-tissue injuries in four cases, fractured humerus and associated soft-tissue injury in one case, and physical neglect and starvation in two cases. Injuries were inflicted by the mother in five instances and by her partner in two cases. In the remaining seven families abuse was suspected but not confirmed (suspected abuse (SA) group). There were 13 families in the no abuse (NA) group with no history of abuse and no direct concern expressed by referrer or Cassel worker about possible abuse to a child.

Demographic data including information about current family circumstances are given in Table 1. The average age of the mothers on admission to hospital in the CA group was 27 years (s.d. 5.1) and in the NA group was 31 years (s.d. 7.1). The average age of mother at first pregnancy was 23.9 years (s.d. 5.7) in the CA group and 23.7 years (s.d. 4.3) in the NA group. More mothers in the CA group were aged 25 years or younger on admission ($P=0.008$, Fisher's exact probability). More children were aged five years or younger at admission in the CA group than in the NA group ($\chi^2=7.34$, d.f.$=1$, $P<0.01$). Almost half the children were six months or younger on admission in the SA group, half were two years or younger on admission in the DA group, and more than half were six years or older on admission in the NA group.

Table 1
Demographic data (including information about current family circumstances) and mothers' histories for the families with ($n=14$) and without ($n=13$) a history of child abuse

	Child abuse group n	No abuse group n
Demographic data		
Age of mother on admission:		
≤25 years	6	1
>25 years	8	12
Age of mother at first pregnancy:		
≤20 years	6	4
>20 years	8	8[1]
Age of children on admission.		
≤5 years	19	11
>5 years	2	12
Sex of children:		
no of boys	10	16
no. of girls	11	7
Mothers currently living:		
alone/no partner	11	5
with partner	3	8
No. of agencies currently involved:		
>2	14	7
≤2	0	6
No. of agencies involved over all time:		
>6	9	2
≤6	5	11
Mothers' histories		
Previous psychiatric diagnosis		
personality disorder	10	4
affective/neurotic disorder	4	9
No. of past psychiatric admissions		
≥1	8	8
0	6	5
History of violence to others	5	2
History of adolescent disturbance	13	7
History of physical abuse as child	8	3
History of sexual abuse as child	5	1
No history of physical or sexual abuse	6	10
Permanent separation from a parent before 18th birthday	9	5
Death of a parent before 12th birthday	3	1
Time spent 'in care' as a child	4	3

[1] One family was childless

More mothers in the CA group lived alone or with no partner ($P=0.007$, Fisher's exact probability). Families in the CA group had more professional agencies involved in their care currently ($P=0.001$, Fisher's exact probability) and over all time ($P=0.02$, Fisher's exact probability).

Problems identified

Violence or potential violence to a child, as well as the passive abuse or neglect of and safety of a child, were defining criteria for the CA group, and consequently all 14 families where such problems exist were included in the CA group. Other problems identified by professionals involved with the families were remarkably similar in both groups.

The most common problem identified by the referrer was that of 'disturbed mother' (22 cases), with the next most common involving the 'parent–child relationship' (21 cases). Less common were difficulties in the 'marital relationship' (eight cases), an identified 'disturbed child' (seven cases), and an identified 'disturbed father' (only four cases).

Problems identified by Cassel workers were established as foci for the family treatment. There was a focus on 'mother's functioning' in 20 cases, on 'mother–child relationship' in 18 cases, on 'parental relationship' in 10 cases, on 'child's functioning' in nine cases, and less commonly on the 'father's functioning' and on 'relating as a family'. Where there was any concern about possible abuse of a child in the family, the safety of the child was of primary importance.

Procedure

This was purely a descriptive study. It did not involve any alterations in the usual clinical practice in the Families Unit. The authors were in continuing clinical contact with the families throughout their admission, KH as manager and senior registrar, RK as consultant psychotherapist, and JS as registrar on the Families Unit.

A 129-item questionnaire was completed on each family by the researcher (clinician). Of the total questions, 10 related to problems identified by professionals involved with the family, 25 concerned current family circumstances, 30 the histories of the parents, 40 examined the qualities of lifetime relationships established by family members and the remaining 24 explored changes made by the family during treatment.

Terms used were described and explained in the questionnaire. The concepts of 'good' and 'poor' relationships were clearly defined. With regard to the problems identified by referrers and Cassel workers, changes made during treatment were coded on a seven-point scale: major negative change (1), important negative changes (2), minimal negative change (3), no change (4), minimal positive changes (5), important positive change (6) and major positive change (7). Change was rated as 'beneficial' if it was coded as 5, 6 or 7 on the scale and as 'no change/worse' if coded as 1, 2, 3 or 4.

Information was gathered from various sources including the family therapist(s), family nurse, daily staff meetings, whole hospital staff meetings, teacher and nurses working with the children in the day unit, observations by the duty/night nurse, meetings with referrers and other professionals involved with the family, and observations from the other families living in close proximity within the hospital. Regular review meetings on the work with each family provided a more formal setting for the gathering of information from workers directly concerned with the family. In this setting transference and countertransference issues were explored and understood. When particular information was not available it was coded as 'not known' on the questionnaire. The information was gathered and coded by KH and JS. To check reliability of ratings, four families were coded on the questionnaire by both workers separately. There was 100% agreement between them on the 65 factual questions. There were minor differences in the coding of the 40 questions on lifetime relationships of family members, and in the coding of the 24 questions on changes made during treatment. These differences did not affect the overall ratings of relationships, and of changes made during treatment, used in this study to compare the groups.

Data analysis

The information collected on the different groups was compared using the χ^2 test with Yates' correction factor. Where the χ^2 test was inappropriate because of small numbers the exact probability test was used (Swinscow, 1983).

Results

The number of fathers admitted to the Families Unit during the period of this study was small (nine in total, three in the CA group and six in the NA group). No useful comparisons between the groups concerning fathers was possible.

The great majority of mothers in the CA group had a previous psychiatric diagnosis of personality disorder, while in the NA group the majority diagnosis was of affective/neurotic disorder. Equal numbers in both groups had previous psychiatric admissions.

The majority of mothers in the CA group had been physically and/or sexually abused themselves as children, in contrast to less than a quarter of mothers in the NA group. Mothers in the CA group had more overt disturbance in their past, and more separations from, or deaths of, parents during their childhood, than mothers in the NA group. There was little difference in their histories between mothers in the DA and SA subgroups.

Four of seven mothers in the DA group, seven of 13 mothers in the NA group, and only one of seven mothers in the SA group remembered at least one good relationship from their childhood. Four of seven mothers in the DA group, six of 13 mothers in the NA group and only one of seven mothers in the SA group established a good relationship with either nurse or therapist during treatment. Six of 13 mothers in the NA group formed a good relationship with other staff and other patients during treatment compared with one of seven mothers in the DA group and no mothers in the SA group.

There was a clear link between the ability to remember a good relationship from childhood and the ability to establish a good relationship during treatment. Of the 12 mothers who recalled at least one good relationship from childhood eight established at least one good relationship during treatment. In contrast, of the 15 mothers who could recall no good relationship from childhood only three established at least one good relationship during treatment ($\chi^2=4.2$, d.f. $=1$, $P<0.05$).

The SA group did very badly on all measures of change during treatment. Beneficial change occurred in one of 16 problems identified by referrers in the SA group compared with change in 12 of 21 such problems in the DA group ($\chi^2=8.2$, d.f. $=1$, $P<0.01$), and in 22 of 39 such problems in the NA group ($\chi^2=9.8$, d.f. $=1$, $P<0.01$). Beneficial change occurred in three of 19 foci established by the Cassel workers in the SA group, compared with 14 of 21 foci in the DA group ($\chi^2=5.6$, d.f. $=1$, $P<0.02$), and 24 of 37 foci in the NA group ($\chi^2=15.7$, d.f. $=1$, $P<0.001$). There was no significant difference between families in the DA group and NA group on either of these measures of change.

Six of 13 families in the NA group, five of the seven families in the DA group, and only one of the seven families in the SA group were in treatment for nine weeks or longer. There was a clear link between the length of admission and the quality of relationships recalled from childhood and established during treatment by mothers. Fifteen families stayed in treatment for eight weeks or less. Mothers in nine of these families had both consistently poor relationships remembered from childhood and observed during treatment. In contrast, mothers in only three of the 12 families who stayed in treatment for nine weeks or longer had both consistently poor relationships remembered from childhood and observed during treatment.

Discussion

The families described in this study were admitted to hospital for a variety of different reasons. They varied in constitution, in size, and in the problems in family functioning experienced. The treatment of some of these families is described briefly by Kennedy (1988).

The 14 families in the CA group were similar in terms of current circumstances and background histories to other abusing families described in the literature. In comparison with mothers in the NA group, those in the CA group were more likely to be younger, without a partner and in contact with multiple professional agencies, as well as to have been abused themselves as children, and disturbed as adolescents. The null hypothesis, that there is no difference between abusing and non-abusing families as experienced in this setting, was rejected.

The major strength of this study is the quality of the information collected and the relevance of this to the lives and daily functioning of these particular families. Although the number of individuals studied is small (80 in total in the 27 families), a psychodynamic perspective on child abuse enhances our understanding of individual cases and also enables agencies working in this area to plan their services with due regard to the needs of their staff (Trowell, 1986). In this setting, where the authors were actively involved clinically with the families throughout their admission, considerable understanding of family functioning developed. An understanding of transference issues, and of countertransference responses, arising during treatment facilitated the actual experiencing of family strengths and pathology. A fuller study of relationships established by family members over time was therefore made possible.

Although neither psychiatric disorder nor patient status appears to bias retrospective recall of early childhood environment (Robins *et al*, 1985), the quality of remembered childhood relationships in this study was understood more as an indicator of current internal object relationships. Mothers from the SA group, where child physical abuse was suspected but not admitted, differed most markedly from mothers in the other groups in terms of the poor quality of their relationships remembered from childhood and observed during treatment. Families from the SA group did worse on measures of change during treatment, and were most likely to remain only a short time in treatment.

Perhaps surprisingly, the seven families where abuse had definitely occurred had better remembered and observed lifetime relationships than the seven families suspected of abuse. They spent longer in treatment and did much better on all measures of change during treatment. These differences confirm, through systematic data collection and analysis, a long-held clinical impression that families who acknowledge the abuse of a child have a greater likelihood of successful treatment. Families in the SA group did not admit abuse. Their failure to recognise the risks to a child in the family was always accompanied by serious concern from professionals involved in their supervision and care.

A major problem in families where abuse has occurred, or is likely to occur, is the absence of a normal parental relationship. This absence usually spans generations. Many mothers in the CA group had experience of permanent separation from a parent during childhood. Few had any relationship with a current partner. A glaring omission from this study is the absence of information on fathers and father figures, in most cases reflecting the absence of any such figure in the families studied.

The quality of relationships established by mothers throughout their lives is identified as a factor within family functioning that may be predictive of ability to engage in treatment, and benefit from it, in cases of child physical abuse. Good relationships remembered by the mother from her childhood increase the likelihood of establishing good relationships during treatment, and improve the chance of having a successful treatment for the family and the abused child.

The above findings await verification on a larger population using standardised measures of the quality of relationships remembered from childhood, observed currently and established during treatment. Such standardised measures, and valid and reliable measures of changes made during in-patient family treatment, are currently being developed as part of a larger research project on the treatment of child physical and sexual abuse in the Families Unit at the Cassel Hospital.

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The Berkeley Group: Ten Years' Experience of a Group for Non-violent Sex Offenders

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During the first ten years of a group started in February 1977 by the Avon Probation Service for the treatment of non-violent sex offenders, many of the offenders have shown a high degree of commitment to the group, and attendance levels have run consistently at over 70%. Of 63 men who came to the group during the ten-year study period, 33 completed their stay at the group, 11 left the group prematurely, and 11 never engaged satisfactorily. The remaining eight were still attending the group at the end of the study period. Of the 55 men whose contact with the group had ended, 36 (65%) had not been convicted of further sex offences by the end of the study period.

Although found guilty of less than 1% of indictable crimes (Home Office, 1986*a*), sex offenders make up 5% of the convicted prison population (Home Office, 1986*b*). The majority serve at least part of their time in prison segregated from other prisoners, to protect them from abuse, threats and actual violence. Under Prison Rule 43, a prisoner, either at his own request or on the instigation of the prison authorities, may be removed from association with other prisoners for the maintenance of good order and discipline or in his own interests (Williams, 1980).

A consideration of the range of sexual offences that come before the courts every year shows that in over half, no violence or coercion is used (Walmsley & White, 1979). The presence or absence of violence, together with other factors such as the age of the victim and previous criminal record of the offender, weigh heavily with the courts in determining the sentence in each case, and are reflected overall in the *Criminal Statistics* published annually by the Home Office. Thus in 1986, of 5400 men convicted of indictable sexual offences, 35% received immediate custodial sentences, 9% received suspended sentences, 15% received probation and 9% conditional or absolute discharge. There were a number of other outcomes such as community service orders and fines. Indecent exposure, as a summary offence, is excluded from these figures, but a custodial sentence follows in less than 5% of those convicted (Howard League Working Party, 1985). As regards reoffending, it has been shown that although the risk in any one year is low, for sex offenders it persists over many years (Soothill *et al*, 1978; Christiansen, 1983).

In 1976, a survey within the Avon Probation Service revealed that probation officers were often uneasy working with sex offenders. They felt uncertain about day-to-day management issues and overall treatment objectives, as well as being unhappy about the lack of supportive supervision. In discussion with a consultant psychiatrist (FGR) who has a special interest in the treatment of sexual deviancy, it was decided to set up a group which could provide a referral point for probation officers who were working with non-violent sex offenders. Once the group became established, it was possible to include a recommendation to attend it in social inquiry reports prepared for the courts (Fox, 1980).

The group meets once every two weeks for one and a half hours, and this paper gives an account of its first ten years.

The group

Because probation is not an uncommon outcome of convictions for non-violent sexual offences, the particular aim of the Berkeley Group was to offer group therapy over an extended period to individuals whose offences were sufficiently grave to make imprisonment a real possibility, whether for the first time or for a further spell. By being able to build such a recommendation to attend the group into social inquiry reports, it was hoped that a probation order would seem a realistic alternative to custody in at least some borderline cases.

There were three rigid criteria that had to be satisfied before such a recommendation was made to the court. Firstly, offenders had to have a minimum of two convictions for sexual offences, in recognition of the fact that the majority of men convicted of a single sex offence never reoffend. Secondly, potential members of the group had to be over 21 years old. The age restriction was introduced to avoid the danger of confirming a young man in a sexual identity or behaviour which was in fact a manifestation of a maturational problem and therefore might be overcome by individual counselling and the passage of time. Thirdly, and most important, was the decision to take only non-violent sex offenders. This criterion was adopted after careful consideration and has been adhered to throughout the ten years. It was felt, and subsequently proved to be the case, that a group made up of paedophiles, men who committed acts of indecency, and exhibitionists had a reasonable prospect of cohering. To introduce into such a group individuals with violent tendencies, might have very well disrupted the therapy and raised anxieties in the group to unacceptable levels. Furthermore, men who had committed violent sexual crimes would almost invariably have been in a position to enter the group only at the end of prison sentences, and this would also have distinguished them from most of the members. However, there is no sharp cut-off point between the use or avoidance of violence, and there were certainly some offenders in the group who had used a degree of coercion or intimidation in some of their offences. In practice, the policy meant that the group did not take any offenders convicted of rape.

In setting up the group it was decided there would be two therapists (CAF, CMW), who would regularly discuss the group process and keep written records of the meetings. Although the therapists have remained unchanged over the ten years, both have taken breaks of up to three months on occasions, and at such times an alternative therapist has been co-opted from within the probation service. This arrangement has worked with very little disruption and does not seem to have had any effect on drop-out rates. Discussion of group issues with a consultant psychiatrist (DAGC) has taken place at two- to three-monthly intervals.

The objectives of the group have been to facilitate discussion of mutual problems, reduce feelings of isolation and abnormality, confront members with the effects of their behaviour on victims, to provide strategies for the management of sexual feelings, and to promote the development of wider social contacts as part of the process of disengaging from the group.

Selection took place at a preliminary interview with the two group leaders. This meeting was used to assess the motivation of the prospective members, provide information about the group, and discuss doubts and anxieties. Six men decided not to participate at this stage and were excluded from the study. However, if the individual was uncertain, it was usually suggested that he commit himself to six sessions and then review. In addition to referrals from within the probation service, there have been five referrals from local consultant psychiatrists. Three attenders have joined the group following lengthy prison sentences, at the suggestion of the prison probation officer. Although when the group was first formed a few offenders had attendance built in as one of the conditions of their probation orders, it was felt that this was undesirable, and that coercion to attend might endanger the group process. For the overwhelming majority of offenders, attendance at the group has been voluntary, despite the fact that they have been on probation orders.

The group meetings have a standard format, beginning with about 15 minutes of informal socialising. The main discussion of the evening lasts one and a quarter hours and the theme may follow a pre-arranged agreement from a previous meeting, arise spontaneously, or be initiated by a particular individual who feels a need to talk about a pressing problem.

Periodically, the therapists review progress with the clients as part of the group business. They do this by considering a checklist of key issues. These are: (a) acknowledgement of the problem; (b) discussion of the build-up to offences; (c) development of 'alternative strategies' when at risk; (d) implementation of these strategies; and (e) development of supports outside the group.

Over the ten years certain general features of the group have become obvious, despite the changing membership. The majority of attenders have shown a high level of commitment to the group. Since the membership is never allowed to exceed ten, the meetings are typically attended by seven or eight offenders. New members have been welcomed and assimilated without disruption. Most men have shown reluctance to be specific about their actual offences, although not to the build-up and circumstances of their offending. The dilemma of wanting to stop offending while at the same time enjoying the actual behaviour has been a recurrent theme of the group discussions. The denial of the effects of offending on victims is repeatedly subjected to scrutiny. The special quality of support that the group provides arises from the recognition of the participants that they are stigmatised by society, but that in the meetings, at least, they can feel and be themselves. Issues raised at the time an individual is preparing to leave the group are often very powerful and may result in discussions lasting over many meetings and involve other members as well as the therapists.

Several of the men convicted of indecent assault on adults or indecent exposure had experienced intercourse with a consenting partner, while the remainder expressed at least some conventional aspirations. There was therefore the hope that therapy would help them towards a better adjustment. By contrast, as was acknowledged in the group, the men convicted exclusively of offences against children outside the family did not change their fundamental sexual orientation through attendance.

Unfortunately, a member sometimes ceases to attend the group without prior discussion. When this happens, the offender's probation officer is contacted so that no misunderstanding can arise; but other than a letter, or sometimes two letters from the group, little pressure is placed on the individual to return.

In addition to the two-weekly group meetings, there is a summer outing for a day and a Christmas drink. At these activities old members sometimes return for a year or two after leaving the group.

Method

Of the 63 men who attended the Berkeley Group at least once between February 1977 and January 1987, 55 had completed their association with the group while eight were continuing to attend. It is the former 55 men who are the subject of this evaluation.

For each individual, the criminal record up to the end of the study was obtained. The offences of each individual were then ordered into those committed before attendance at the group, those committed while attending, and those committed after leaving.

Because, when the group was set up, the over-riding concern had been to provide a therapeutic programme for sex offenders as a resource to the Avon probation service, there was no attempt to establish a control group of offenders who received no intervention or a different type of intervention. Nevertheless, despite methodological shortcomings, it was felt important to evaluate the work of the group both because of its endurance over a long period and because of a lack of research in this field (Mendelson *et al*, 1988). Consequently, the 55 men were divided into three subgroups, according to whether or not they had fulfilled the aims of the programme to the satisfaction of the group leaders, and in this way those who failed to engage and those who defaulted could be compared with those who completed therapy.

A total of 33 men (the 'completion group') completed their attendance and left at a time planned with the leaders of the group. Of these, three committed relatively minor sex offences within a few months of first attending the group. The leaders permitted the men to continue participating until they appeared in court. The final outcome in each case was that the probation order was continued, so that attendance was maintained almost without interruption.

Eleven men (the 'default group') attended for substantial periods (range 3–19 months), but ultimately left the group against the advice of the leaders, or in the case of five individuals by reoffending and being taken into custody. A further eleven men (the 'non-engagement group') scarcely made contact with the group, attending only once, twice or three times.

Results

Table 1 records the most serious sexual offences of the 55 men at the time of their referral to the group.

Table 2 summarises details of the criminal records of the men before and after their referral to the group, together with information about their lengths of stay in the group and time elapsed since discharge.

A comparison of all three subgroups using a Kruskal-Wallis analysis showed that the median number of sex offences committed before attendance was equivalent for the 'completion' and 'non-engagement' groups but was significantly raised in the 'default' group. Similarly, there were significant differences in the median number of other (i.e. non-sexual) offences committed before attendance and again the 'default group' has the higher value.

A χ^2 test showed subgroup differences in the number of individuals who had served prison sentences before attending the group, and in this case the 'non-engagement group' had a significantly lower proportion of previously imprisoned offenders.

Table 2 also shows that the distribution of individuals between subgroups who went on to commit at least one further sexual offence was not consistent with a chance hypothesis ($\chi^2=9.17$, d.f. $=2$, $P<0.05$). This result was caused by an elevated proportion of reoffenders in the 'default group'.

There was also a significant relationship between a previous prison sentence and sex offence after leaving the group. Thirteen of the 19 reoffenders had served a prison sentence compared with 13 of the 36 men who did not reoffend ($\chi^2=3.99$, d.f. $=1$, $P<0.05$).

Of the 26 men who had served previous prison sentences, those who completed attendance at the group were significantly less likely to commit further sex offences than those in the combined 'default' and 'non-engagement' groups ($\chi^2=4.06$, d.f. $=1$, $P<0.05$) (Table 3). The post-group offending behaviour of the 29 men who had not served previous prison sentences was not significantly different, whether the programme had been completed or not (Table 3).

Table 1
Most serious offences for which 55 men were convicted before attending Berkeley Group

	'Completion group' ($n=33$)	'Default group' ($n=11$)	'Non-engagement group' ($n=11$)
Gross indecency with a child	11	2	3
Indecent assault on a woman	7	5	2
Indecent exposure	5	4	4
Buggery	5	–	–
Indecent assault on a man	3	–	2
Unlawful sexual intercourse	1	–	–
Incest	1	–	–

Table 2
Criminal records before and after therapy and attendance patterns of 55 men referred to the Berkeley Group

	'Completion group' ($n = 33$)	'Default group' ($n = 11$)	'Non-engagement group' ($n = 11$)	Statistical comparison
Mean (range) age: years	38.6 (21–67)	31.1 (21–51)	31.8 (23–59)	NS
No. with one or more convictions as a juvenile	7	3	1	NS
Mean no. of convictions for sex offences before attending group	3.7 (2–21)	6.6 (3–29)	2.8 (2–4)	
Median no. of convictions for sex offences before attending group	3	5	2	Kruskal-Wallis $\chi^2 = 9.82$** d.f. = 2
Median (range) no. of convictions for other offences before attending group (range)	1 (0–8)	5 (0–19)	0 (0–6)	Kruskal-Wallis $\chi^2 = 8\ 97$* d.f. = 2
No. who had served one or more prison sentences before attending group	16	8	2	$\chi^2 = 6.61$* d.f. = 2
Median (range) length of stay in group: months	22 (4–51)	6 (3–19)	–	Mann-Whitney $U = 56.5$***
Median time since leaving the group: years	4.9	4.8	4.1	NS
No. who committed further sex offences after attending the group	9	8	2	$\chi^2 = 9.17$* d.f. = 2

*$P < 0\ 05$, **$P < 0.01$, ***$P < 0.001$

The median number of previous sex offences and previous other offences were 4.0 and 4.0 respectively in the reoffending group and 2.5 and 0.0 respectively in the non-reoffending group. The differences between both are significant (sex offences: Mann-Whitney $U = 215$, $P < 0.05$; other offences: Mann-Whitney $U = 123.5$, $P < 0.001$). This finding supports the hypothesis that number of previous offences is a strong indicator of subsequent offending behaviour.

Finally, six of the paedophiles had at some time committed offences with family members, in one case incest and in the remainder indecent assaults. Of these offenders, two committed further sexual offences, both outside the family.

Discussion

The evaluation of the effectiveness of the Berkeley Group in preventing reoffending has been conducted in terms of three subgroups, only one of which, containing 33 of the 55 referrals, completed the therapeutic programme. The median length of stay of nearly two years is the bare statistical indicator of sustained commitment to the group.

Comparing the 'completion group', the 'default group' and the 'non-engagement group' reveals important differences. The 'default group' has members with a significantly higher number of sexual and other offences (Table 2). In other words, the 'default group' contains the most troublesome recidivists. This statement should be qualified by noting that 11 members of the 'completion group' had committed four or more sex offences (range 4–21), and six of these 11 had committed other offences as well. The 'non-engagement group' has members with significantly fewer previous prison sentences than the other two subgroups (Table 2). This finding suggests that the 'non-engagement group' contains the least serious offenders. It seemed from remarks that members of the 'non-engagement group' made before leaving prematurely, that many

Table 3
Reoffending (sexual offences only) in 26 men who had served prison sentences before attending the group and 29 men who had not

	Previous prison sentence[1]		No previous prison sentence[2]	
	'Completion group'	'Default' & 'Non-engagement groups'	'Completion group'	'Default' & 'Non-engagement groups'
Sex offence committed after leaving group				
yes	5	8	4	2
no	11	2	13	10

1 $\chi^2 = 4.06$, d.f. = 1, $P < 0\ 05$.
2 $\chi^2 = 0$, d.f. = 1, NS.

perceived their problems as less severe than those of other group members.

The most important outcome measure is the statistically significant difference in the reoffending rate of the 'completion' as compared with the 'default' group. It is not possible to attribute this finding to therapeutic effects alone: such an unambiguous conclusion would only be possible if the 'completion' and 'default group' were comparable in other respects. However, as we have shown, the 'default group' had worse previous criminal records, and this itself is a predictor of poor outcome. Furthermore, if the 'default group' is taken together with the 'non-engagement group' and compared with the 'completion group', the statistically significant difference in offending rate is lost.

There is one unambiguous piece of evidence of the therapeutic effectiveness of the group. Of those 26 men who had served prison sentences before attending the group, significantly fewer from the 'completion group' reoffended than from the other two subgroups combined (Table 3).

Is it justifiable to conclude that there is an optimal time for referral to a group such as this? On the basis of the characteristics of the 'non-engagement group' and the 'default group', one might argue that, on the one hand, offenders who have been shaken by court appearances and, on the other hand, offenders who show the incorrigibility of recidivists are not ideal candidates for the kind of non-coercive therapy offered by the Berkeley Group, but this argument depends on the benefit of hindsight. There is no reason for not considering group therapy for a sex offender simply because the prognosis is poor, although any such recommendation must be argued for when he appears before the court. Nevertheless, despite the unsurprising confirmation that recidivism predicts a poor outcome, it has been shown that of those group members with previous prison sentences who completed therapy, a statistically significant better outcome is achieved (Table 3). Six members of the 'completion group' with four or more previous sex offences did not reoffend after leaving the group. Five of them had served prison sentences before attending the group. These six offenders had been away from the group for an average of 4.75 years (range 3.25–7.5 years).

In view of the fact that the offenders with the worst criminal records are most likely to reoffend, one may wonder whether sanctions to compel attendance should be sought. Obviously such an approach would be possible by making attendance at the group a condition of probation. Although compulsion cannot guarantee successful psychotherapeutic work, it does not preclude it either. Nevertheless, we took the view that in highly stigmatised individuals, coercion would be likely to be counterproductive, exaggerating passive resistance and hostility. Certainly the commitment of the majority of referrals to the group was very striking, although generally the defaulters fell into two categories, those with the least severe and those with the most severe problems.

It is perhaps surprising that in ten years only six of the offenders had committed assaults on family members. However, since the conclusion of the study, dramatic changes have taken place. Thus, at the time of writing, three out of nine of the members of the Berkeley Group and approximately half of the members of a second group, started in July 1987 to cope with the greater number of referrals, had assaulted female relatives or step-daughters. Because these individuals usually have to wait several months before appearing in court, some of them have attended the groups while on remand, a development which is felt to be useful because denial can be confronted at an early stage. Needless to say this does not prejudice the court proceedings, and some offenders have been imprisoned in due course. These new trends are important, but it would be premature to discuss them in detail in this paper.

Finally, although the focus of this paper has been on client outcomes, the group's own ten-year survival in an area of underprovision deserves comment. The leaders' commitment was obviously essential. They maintained the group's basic structure and availability over the years and, in so doing, gave a strong message about how they viewed their clients' worth. Both outside consultants (DAGC and FGR) felt that the leaders provided a remarkably consistent level of informed acceptance, which seemed crucial to the group's ability to combine support and confrontation effectively.

Conclusions

The task of deterring, much more reforming, sex offenders is a difficult one. This paper records a sustained attempt to achieve such ends through the use of group therapy with non-violent offenders. Whether as time passes the offenders who completed the therapeutic programme will continue to diverge from the defaulters in terms of reoffending is uncertain, but might form the basis of a further study. Long-term support for offenders, perhaps to be used intermittently when adverse life circumstances make the risk of reoffending higher, might be a useful development in the future.

Undoubtedly the growing recognition of the problem of child sex abuse within the family will change the sort of work described in this paper. The way in which the role of the probation service should be integrated with the work of other agencies is a major challenge for the future.

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Intoxicated by Water

Polydipsia and Water Intoxication in a Mental Handicap Hospital

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A cross-sectional survey of the drinking habits of 877 mentally handicapped in-patients revealed 31 patients (prevalence 3.5%) who, in the opinion of nurses, drank five litres or more daily. Low urine specific gravity was a less useful indicator of polydipsia. Polydipsia appeared to be significantly associated with a borderline level of handicap and with a diagnosis of schizophrenia, autism or severe personality/behaviour disorder. Of five cases of water intoxication associated with polydipsia, one was fatal. In two cases excess drinking improved with increased neuroleptic medication. Lithium and demeclocycline were used in two cases to prevent hyponatraemic episodes.

Primary polydipsia or compulsive water drinking is a syndrome in which a person drinks large amounts of water for obscure reasons unrelated to normal thirst or homeostatic needs (Raskind & Barnes, 1984). The normal adult can excrete 15 litres of water a day, but if intake temporarily exceeds this rate (e.g. 3 or 4 litres drunk over 20 minutes) retention can occur. There may also be abnormal physiological mechanisms which lead to water retention, including the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) (Bartter & Schwarz, 1967). Rapid development of hyponatraemia and hypo-osmolality leads to water intoxication, an encephalopathy presenting as restlessness, confusion, lethargy, nausea, diarrhoea, vomiting, muscle twitching, ataxia, convulsions, seizures, stupor, coma and occasionally death (Rowntree, 1923).

Primary polydipsia has been associated with a variety of psychiatric diagnoses and thought to be relatively benign until the 1970s. It was first recognised in schizophrenia (Sleeper & Jellinek, 1936; Barahal, 1938) and later associated with neurosis and personality disorder (Barlow & De Wardener, 1959) and organic brain syndromes (Bewley, 1964; Stevko *et al*, 1968). Reports were published of the association of water intoxication with acute psychosis, both schizophrenic (Fowler *et al*, 1977; Rosenbaum *et al*, 1979; Kramer & Drake, 1983) and affective (Raskind *et al*, 1975; Zubenko *et al*, 1984).

In recent years evidence was accumulated for a variety of long-term complications of chronic polydipsia. As well as recurrent episodes of water intoxication, these include bowel and bladder dilatation (Viewig *et al*, 1984), hydronephrosis, renal failure, malnutrition, projectile vomiting, cardiac failure and dementia (Blum *et al*, 1983), hernias and incontinence (Delva & Crammer, 1988), and spinal fractures secondary to calcium loss (Delva *et al*, 1989).

Although polydipsia and water intoxication have been studied in psychiatric populations, there have been no systematic surveys in mentally handicapped populations and few case reports (Mendelson & Deza, 1976; Noonan & Anath, 1977; Jose & Perez-Cruet, 1979). We describe a cross-sectional survey of polydipsia in mentally handicapped patients followed by case histories and management of water-intoxicated patients.

Method

The population comprises the 877 in-patients of a large Victorian hospital on the outskirts of north London. All have been diagnosed as mentally handicapped with a range of ability from profound handicap to borderline average intelligence. Included in the population is the regional secure unit for mentally handicapped patients. Patients over 70-years-old comprise 20% of the population and the proportion of males in this group is 37%. Of those aged under 70 years, males comprise 55% of the population.

The sample is defined as those patients who were estimated by staff to drink five litres or more per day (measured in cups) and/or who went to extraordinary lengths to drink fluid. The latter clause is to include those patients who had limited access to water, but who would exploit any opportunity (e.g. drinking avidly from bath or urinal) so that the amount ingested was very hard to estimate. The whole hospital was screened for urinary specific gravity to identify those with a specific gravity of less than 1.008 on three occasions and to determine the usefulness of this measure as an aid to detecting polydipsic patients.

Exclusion criteria were renal disease, diabetes mellitus, diabetes insipidus, and use of diuretics or lithium. An early-morning urinary osmolality of greater than 300 mosm/kg with a serum sodium of less than 148 mmol/l was taken to exclude diabetes insipidus. Patient consent or written consent of the nearest relative was obtained for the following investigations: serum urea and electrolytes, glucose, creatinine, osmolality, early morning urinary sodium and osmolality and diurnal weighing (08.00 and 20.00 h).

A group of non-polydipsic patients (non-drinkers) were matched with drinkers for ward, age, sex, weight and level of handicap, then had daily fluid intake assessed and were also weighed diurnally; more invasive investigations were not performed in this group for ethical reasons. We interviewed each polydipsic patient and a staff member who knew the patient well over a long period. We examined case notes to determine level of handicap, psychiatric, medical and family history, the course of the person's development or illness, the response to medication and current medication. We inquired about smoking, eating and behavioural habits. We made an assessment psychiatric diagnosis based on all information available. We assessed personality guided by a standardised personality assessment (Mann *et al*, 1981) which has been used previously for mentally handicapped patients (Ballinger & Reid, 1987). Where possible patients were also interviewed for their views on the reasons for their drinking so much. Statistical analysis was performed using the fourfold χ^2 test with Yates' correction for continuity.

With respect to water intoxication, 'cases' were defined by neurological symptoms in association with low serum sodium and/or osmolality, and a history of recent polydipsia, with no other cause for the neurological symptoms or hyponatraemia being apparent.

Results

Of 877 patients we found that 31 (3.5%) frequently drank over five litres a day without an apparent physical cause, or went to extraordinary lengths to obtain water. Eight drinkers had been excluded on physical grounds (two with diabetes mellitus, five taking lithium, and one taking diuretics). Of the 31 patients, six had repeated low urinary specific gravity, but only one patient was identified by specific gravity alone and subsequently discovered to be drinking excessively. It was impossible to test the urine of 50 (6%) patients because of difficulties with patient cooperation or incontinence. Another 33 (4%) patients had a low specific gravity as a result of age (29), renal disease (2) or lithium (2).

There were 21 men and 10 women, whereas the sex ratio is almost 1:1 in the total hospital population ($\chi^2 = 3.02$, $P < 0.1$). The average age of drinkers was 42 (range 24–84 years), compared with an average age of the whole hospital population of 49 years. A significant association with borderline handicap was found, whereas absence of polydipsia was found to be associated with severe mental handicap ($\chi^2 =$ 29.94 and 4.16, $P < 0.001$ and 0.05 respectively) (Table 1).

Table 1
Degree of mental handicap in polydipsic sample compared with the general hospital population

Level of handicap (ICD-9)	IQ	Drinkers (*n* = 31) no.	(%)	Hospital population (*n* = 877) no.	(%)
Borderline[1]	70–85	7	(22)	30	(3)
Mild	50–69	9	(29)	149	(17)
Moderate	35–49	6	(19)	201	(23)
Severe[2]	20–34	7	(22)	368	(42)
Profound	<20	2	(6)	75	(9)
Unspecified		0	(0)	54	(6)

1. $\chi^2 = 29\ 94$, d f = 1, $P < 0\ 001$
2. $\chi^2 = 4\ 16$, d.f. = 1, $P < 0\ 05$

There was no drinker newly diagnosed as having diabetes mellitus or diabetes insipidus. Serum sodium of drinkers ranged from 127 to 140 mmol/l with a median of 135 mmol/l (normal range was 136–148 mmol/l). However, it should be borne in mind that blood samples were taken in the early morning when drinking behaviour would have least effect (although this enabled us to use the results to exclude diabetes insipidus). Serum creatinine of drinkers ranged from 35 to 78 μmol/l (normal range 60–120 μmol/l)

Drinkers had an estimated daily fluid intake ranging from 5 to 16 litres (median 6 litres) whereas matched non-drinkers had a range of 1–3.5 litres (median 2 litres). The weight gain of drinkers over 12 hours ranged from none to 7.5 kg (median 1.5 kg) and of matched non-drinkers from none to 2.5 kg (median 0 kg). Twelve of 31 drinkers had a weight gain of over 2 kg compared with 3 of 31 non-drinkers ($\chi^2 = 5.63$, d.f. = 1, $P < 0.02$).

In comparing the psychiatric diagnoses on the hospital register of drinkers with non-drinkers, significant associations with polydipsia were found for schizophrenia ($\chi^2 = 7.32$, $P < 0.01$), severe personality/behaviour disorder ($\chi^2 = 6.73$, $P < 0.01$) and autism ($\chi^2 = 18.94$, $P < 0.001$). The

Table 2
Psychiatric diagnosis of the polydipsic sample compared with the rest of the hospital population

Psychiatric diagnosis	Drinkers (*n* = 31) Assessment diagnosis no	(%)	Drinkers (*n* = 31) Register diagnosis no.	(%)	Hospital population (*n* = 877) Register diagnosis no.	(%)
Schizophrenia[1]	10	(32)	8	(26)	87	(10)
Manic-depressive psychosis	1	(3)	1	(3)	7	(1)
Past psychosis	2	(6)	2	(6)	27	(3)
Neurosis	2	(6)	1	(3)	11	(1)
Personality/ behaviour disorder (severe)[2]	6	(19)	7	(23)	73	(8)
Autism[3]	6	(19)	5	(16)	22	(2)
Dementia	0	(0)	0	(0)	35	(4)
No psychiatric[4] diagnosis	4	(13)	7	(23)	615	(70)

1 $\chi^2 = 7\ 32$, d f. = 1, $P < 0\ 01$.
2 $\chi^2 = 6\ 73$, d f = 1, $P < 0\ 01$
3 $\chi^2 = 18\ 94$, d f = 1, $P < 0\ 001$.
4. $\chi^2 = 23\ 42$, d f = 1, $P < 0.001$

Table 3
Personality assessment in polydipsic patients

Personality type	Grade I[1]	Grade II[2]	Total (grade I/II) ($n=31$)
Self-conscious	1	0	1
Schizoid	2	0	2
Paranoid	0	1	1
Cyclothymic	4	0	4
Obsessional	5	0	5
Anxious	6	0	6
Asthenic	2	0	2
Sociopathic	0	1	1
Explosive	3	1	4
Hysterical	1	1	2
'Stable'	3	0	3

1. Grade I, personality can be matched by one of the types.
2. Grade II, personality unusual or handicapping.

validity of the register diagnosis was largely supported by our assessment diagnosis (Table 2). Three men had a history of sexual deviation and one woman had a history of bulimia and anorexia.

Personality types were variable, although cyclothymic, obsessional and anxious personalities were most common, and together accounted for almost half the sample. In those four patients with personality disorder who showed several personality types, the dominant type was recorded (grade II) (Table 3).

Of those 18 patients able to discuss their drinking, 12 admitted they drank a lot; only six said they were thirsty and six said they had a dry mouth. Of those 11 patients who could discuss a reason for drinking excessively, eight did not know why, two said they had a dry mouth, and one said it was a habit.

Of the 31 cases, 15 smoked cigarettes, 11 tended to bolt food quickly, five had pica, nine had repeated self-injury, eight had marked stereotypic movements, and six were generally overactive. Of medical diagnoses recorded, epilepsy was by far the most common and was noted in seven of the drinkers. It is interesting that none had Down's syndrome, although these comprise 8% of the hospital population.

Case histories of water intoxication

In a retrospective survey of the mental handicap population of 877 patients over three years, we knew of nine people who apparently developed water intoxication. Two were not polydipsic and had a clear underlying medical cause (carbamazepine in one case and cardiac failure with thiazide diuretics in the other). The other seven patients were polydipsic at the time of apparent water intoxication. Of these seven, three had chronic polydipsia and had their first episode of water intoxication when being treated with either carbamazepine or chlorpropamide. In the other four patients with episodic polydipsia, water intoxication was in each case associated with psychotic or disturbed episodes and all four were heavy smokers. Of the seven patients, two did not have sodium levels recorded. The five with documented hyponatraemia, satisfying the criteria of 'caseness', are described.

Case 1

A 58-year-old woman with a moderate mental handicap has a 20-year history of schizophrenia as evidenced by auditory hallucinations, incongruous affect, bizarre mannerisms and regression. Her excessive water drinking was noted at the age of 44 years and thought to be due to mild diabetes, which was managed by diet alone. Over the last 12 years she has had several episodes of water intoxication, once with a serum sodium as low as 116 mmol/l, but on each occasion the vomiting, fits and stupor were initially thought to be due to hypoglycaemia or a cerebrovascular accident. She was prescribed phenytoin following one episode described as atypical epilepsy. It is possible that she drinks in response to hallucinations, as nurses have noticed that in periods when she drinks excessively, she talks to an imaginary person and looks upwards for no obvious reason. Furthermore, this excessive drinking and mental disturbance decline with an increase in her maintenance oral haloperidol.

Case 2

A 53-year-old man has an IQ of 38 following brain damage from meningitis in infancy. There is a family history of affective disorder. Over 18 years he has had several clearcut episodes when he is apathetic, anorectic, sleepless, loses weight and wants to die. At other times he has periods when he is restless, garrulous, oblivious to all concerns, hoards rubbish and wants to wander off to sleep out of doors. He drinks tea copiously, smokes compulsively and self-inflicts skin injuries.

He has had two periods of acute confusion with hyponatraemia. The first episode (serum sodium 125 mmol/l) was attributed to thiazide diuretics, which were discontinued. The second occurred during a hypomanic episode, when he was also drinking tea excessively and had been treated with chlorpromazine (300–400 mg/day) over three weeks. On this occasion he wandered off and when found the next day, he was clinically dehydrated with the following biochemistry: serum sodium 122 mmol/l, potassium 3.3 mmol/l (normal range 3.5–5.5), bicarbonate 25 mmol/l (normal range 22–32), urea 10.6 mmol/l (normal range 2.5–6.5), creatinine 105 μmol/l (normal range 60–100), serum osmolality 264 mosm/kg (normal range 270–285), urine osmolality 581 mosm/kg (normal range 40–1400), urine sodium 268 mmol/l, urine potassium 50 mmol/l.

After hospital care his urea returned to normal but he remained hyponatraemic with normal chest X-ray, glucose tolerance test, adrenal-stimulating hormone test, porphyrins, thyroid function and computerised tomography (CT) scan. Chlorpromazine was initially assumed to be responsible for his hyponatraemia, and haloperidol (5 mg/day) was substituted, but his serum sodium remained at 125 mmol/l and he continued drinking excessively. When demeclocycline (300 mg q.d.s.) was added his sodium rose to 130 mmol/l.

With early treatment of his psychotic episodes and monitoring of his fluid intake, there have been no further episodes of confusion and his serum sodium is within normal limits.

Case 3

A 51-year-old menopausal woman with an IQ of 47 was deserted in early childhood by an unstable mother. She has a brother who is itinerant, aggressive and had repeated psychiatric admissions for unspecified psychosis and self-neglect. She is described as the ward bully and has always been touchy, disgruntled and explosive, and has a diagnosis of personality disorder. She has periods of self-injury and is always greedy for sweets and cigarettes. She has had several brief depressive episodes, one with marked hysterical features. Depot flupenthixol decanoate had been prescribed in increasing dosage for years with little behavioural change. As she had never been actively psychotic, the fortnightly intramuscular dose of flupenthixol decanoate was reduced over three months from 100 mg to 20 mg. The antidepressant lofepramine (140 mg/day) was continued over this time.

She was declared fit to go on holiday and that afternoon became confused, vomited, and had a swollen face and a distended abdomen. She then became rigid, cyanosed and comatose and was thought to have a myocardial infarct or a cerebrovascular accident. Investigations showed: serum sodium 118, potassium 3.3, bicarbonate 27, urea 3 mmol/l. Plasma osmolality rose from 252 to 298 mosm/kg, and urine osmolality rose from 153 to 467 mosm/kg with water restriction over 24 hours. She had normal lumbar puncture, electrocardiography (ECG), full blood count, calcium, thyroid function, liver-function test, skull X-ray and CT scan.

When questioned in detail later, staff recalled that she was always at the tap. The depot flupenthixol and lofepramine were suspended. Over the next ten months she had four further episodes of water intoxication with serum sodium levels ranging from 113 to 123 mmol/l. After fluid restriction and a return to normal serum sodium levels she would become depressed and suicidal and did not eat or smoke. She would then drink large volumes rapidly from the toilet and if prevented, became tense and persisted in anal poking and self-mutilation. The extent of her drinking was monitored by daily weighing and she often needed close supervision. With no response to oral antipsychotics and antidepressants, electroconvulsive therapy (ECT) was considered but her water seeking reduced dramatically when depot flupenthixol (40 mg i.m. fortnightly) was restored. Lithium (800 mg *nocte*) was also prescribed primarily for the effect of increasing free water clearance. She remains on lithium and flupenthixol decanoate with no further episodes of intoxication.

Case 4

A 29-year-old man had a normal birth and early milestones. At 18 months infantile convulsions developed and were followed by regression and loss of speech. The subsequent diagnosis of disintegrative psychosis and childhood autism was made. In adolescence he adopted rituals which centred on touching the breasts of women followed by repeated self-mutilation of his eyes. Cataracts developed and despite a series of operations he remained blind in one eye. He continued to produce striking but stereotyped paintings of eyes or severed heads but his speech remained at the four-year level.

At 23 he developed hypertension which was treated with nifedipine. Because of his high anxiety level he remained on antipsychotics with antidepressants when required. At 26 he developed his first episode of water intoxication when he drank large amounts from unusual places, and had hyponatraemia (121 mmol/l) which was thought to be exacerbated by thiazide diuretics and these were discontinued.

His second episode of hyponatraemia (116 mmol/l) was three years later and resolved within 24 hours with fluid restriction. Ten days later he was observed to be drinking water excessively again, then became abusive and tense for several days with the sudden onset of vomiting, diarrhoea and coma. His medication at the time was fluvoxamine (100 mg *nocte*), chlorpromazine (100 mg t.d.s.), atenolol (50 mg *mane*), and prazosin (2 mg t.d.s.). As several empty aftershave bottles were found nearby, self-poisoning was suspected but alcohol was not detected. Serum sodium was 126, potassium 3.2, bicarbonate 22, urea 2.2, and glucose 9.4 mmol/l. Soon after he had a respiratory arrest and became hypotensive. He was resuscitated and given 50 mmol of intravenous sodium bicarbonate and a two-litre infusion of normal saline overnight, but became hypernatraemic (151 mmol/l) with a flaccid tetraplegia. Central pontine myelinolysis (Laureno, 1983) was suspected. A CT scan showed diffuse cerebral oedema and obliteration of the ventricles. He died two weeks later and the post-mortem examination showed total infarction of the cerebral cortices, pons, medulla and cerebral hemispheres, thought to be due to anoxia following his respiratory arrest.

Case 5

A 41-year-old man has an IQ of 41 and a history of epilepsy from the age of two years, with aggressive outbursts and petty thieving from adolescence. When his mother died he was admitted to a centre for epilepsy, where he had confused episodes but no seizures. He began setting fires, usually under the influence of alcohol, and was detained in a mental handicap hospital for close supervision. With each attempt at a hostel rehabilitation programme, he became temperamental, threatened arson, chain smoked and carried a jar of coffee and powdered milk in his pocket to make drinks every five minutes. He had what appeared to be recurrent cerebrovascular episodes until it was clear that he was drinking water to the stage of unsteady gait and slurred speech.

Investigations showed a serum sodium level of 126 mmol/l with normal urea, creatinine, calcium, urinary sodium, fasting lipids, thyroid function, glucose tolerance test and chest X-ray. As he has had no epileptic fits for five years and no other physical cause has been found for his hyponatraemia, carbamazepine is being gradually suspended, and his fluid and cigarette consumption is supervised.

Discussion

Our findings suggest that polydipsia and water intoxication are not uncommon among mentally handicapped patients in long-term hospital care. The prevalence rate for polydipsia of 3.5% compares with a range of 6.6% in 239 patients (Jose & Perez-Cruet, 1979) to 17.5% in 241 patients (Blum *et al*, 1983) found in surveys of American state psychiatric institutions. Our lower rate may reflect an association with mental illness and the fact that not all mentally handicapped patients are mentally ill, or it may reflect the higher level of drinking required to be included as polydipsic in our study. Jose & Perez-Cruet defined polydipsia as simply excessive drinking based on nurses' and patients' estimates, and did not state whether medical causes had been excluded. Blum used a subjective assessment to detect those drinking more than four litres a day with or without low urine specific gravity and excluded physical causes.

In view of the difficulties of obtaining satisfactory measures of drinking, the difference in prevalence rates is not surprising. We found that screening patients for a low specific gravity was not a sufficiently valid method for detecting polydipsia and added little to ordinary nursing observation. For those patients needing close supervision of their drinking, weighing appeared a useful non-invasive guide to water retention and hyponatraemia (Delva & Crammer, 1988), as in our case 3.

The group found to be polydipsic were heterogeneous in terms of psychiatric diagnosis and personality. Individually significant associations were found with schizophrenia, autism, and personality disorder. These associations were not predicted, appearing only in a *post hoc* analysis, therefore repetition of these findings is necessary before they can be accepted as proven. Despite this qualification, the association with autism appears highly significant. It should also be noted that having excluded patients taking lithium from our study on the grounds of its effects on water excretion, we have reduced the likelihood of finding polydipsic patients with affective disorder. In psychiatric patients, water intoxication has been found to be associated with psychosis in 80–85% of cases (Ferrier, 1985; Zubenko, 1987) whereas case descriptions of patients found by physicians to have compulsive polydipsia suggest polydipsia is more evenly spread among psychiatric diagnostic groups (Barlow & De Wardener, 1959; Chinn, 1974; Singh *et al*, 1985).

The sex ratio is the inverse of that expected from previous studies in psychiatric patients: 68% of drinkers were found to be male in our study, as opposed to 78% female (Barlow & De Wardener, 1959) and 67% female (Jose & Perez-Cruet, 1979). In our total hospital population, 55% of in-patients with psychosis are male and 66% of in-patients with autism are male. As polydipsia appears to be associated with autism and psychosis, this may explain the reversed gender trend.

The findings relating to pica and bolting of food were unfortunately uncontrolled, but in themselves are suggestive that, at least in some patients, the polydipsia being observed may be part of a general tendency to ingest.

The clinical significance of polydipsia is highlighted by the case histories of patients with water intoxication. One of the five cases was fatal. This compares with a finding in psychiatric patients that water intoxication is associated with a mortality of 10% over two years (Hariprasad *et al*, 1980). Each of our five cases involved initial misdiagnosis. Diagnosis may be especially difficult in mentally handicapped patients among whom epilepsy is common, as a frequent presentation of water intoxication is a generalised seizure (Singh *et al*, 1985). In the event of late-onset seizures, especially if the patient has recently been more disturbed, it is advisable to obtain a drinking history, and check electrolytes and osmolalities. Anticonvulsants may not be needed, and carbamazepine may compound the problem.

We found no case of water intoxication to be attributable to antipsychotic or antidepressant medication and in two cases (1 and 3), increasing antipsychotic medication appeared to lead to a reduction in water drinking and an end to episodes of water intoxication. It is interesting that Shen & Sata (1983) also observed that a number of episodes of water intoxication occurred among polydipsic patients when their neuroleptic dosage was decreased. Although psychotropic medication is associated with SIADH, so is psychosis, and as Sandifer (1983) points out, psychotropic medication is often falsely blamed for SIADH. He recommends that the causative role of medication should be documented by a rechallenge or by a water-loading test. If a patient develops water intoxication on a newly prescribed (within one month) psychotropic medication, advice is to check sodium, osmolality and ADH levels, and if other physical causes are excluded, to change the drug with a later rechallenge. However, if such a person is drinking excessively, the most probable cause of both water intoxication and polydipsia is inadequately treated psychosis.

If the hyponatraemia cannot be controlled, we would consider adding demeclocycline (Forrest *et al*, 1978; Nixon & Chin, 1983; Sandifer, 1983; Goldman & Luchins, 1985) or lithium (White & Fetner, 1975) to promote free water clearance (cases 2 and 3). Both demeclocycline and lithium act as ADH antagonists at the level of the renal tubule, causing nephrogenic diabetes insipidus and free water excretion (Shrier, 1985). On the other hand, carbamazepine (Perucca *et al*, 1978), thiazide diuretics (Raskind, 1974; Fowler *et al*, 1977) and chlorpropamide (Smith & Clark, 1980) all potentiate ADH by renal or central mechanisms (Shrier, 1986) and can exacerbate hyponatraemia (cases 2, 4 and 5). Hence it seems wise to avoid these medications in polydipsic patients. In addition, nicotine, and thus smoking, induce inappropriate ADH secretion: we therefore followed the advice of Chin *et al* (1976) in restricting cigarettes in those patients with repeated low sodium levels.

In chronic and episodic drinkers, close chaperoning and water restriction should be carried out if serum sodium levels fall below 127 mmol/l, or when the person gains 7% of his morning body weight by the evening (Delva & Crammer, 1988).

There have been many hypotheses advanced to explain the occurrence of polydipsia and the current study does no more than many others to support or refute these explanations. However, our clinical observation in conducting this study suggests that many of the most polydipsic patients were particularly anxious while exhibiting their polydipsic behaviour. If polydipsia is an idiosyncratic response to anxiety, this may explain its association with a variety of psychiatric illnesses. Lee *et al* (1989) have proffered the hypothesis that it is secondary hyponatraemia that is anxiolytic and described a drinker who regularly induced a pleasurable state of altered consciousness. However true this may be of some patients, it cannot explain the occurrence of polydipsia in patients who do not become hyponatraemic. Other explanations have been in terms of family modelling (Kulkarnia *et al*, 1985), and various neurochemical explanations of water drinking to the point of intoxication, including a possible hypothalamic defect (Hobson & English, 1963), a primary inappropriate secretion of ADH (Khamnei, 1984), a reset osmostat (Hariprasad *et al*, 1980; Zerbe *et al*, 1980; Goldman *et al*, 1988) and a hyperdopaminergic state (Smith & Clark, 1980; Illowsky & Kirch, 1988).

We suggest a general explanation of these phenomena might run as follows:

(a) Polydipsia may be viewed as a learned behaviour, which depends on:
  (i) availability of fluid to drink
  (ii) a persistent anxiety, perhaps relating to feelings of emptiness
  (iii) a sense of partial and temporary relief gained from the physical sensation of drinking
  (iv) a lack of other, more effective methods (available to the patient) of relieving this anxiety.

This explanation is not necessarily incompatible with a neurochemical theory of polydipsia.

(b) Water intoxication may occur either when polydipsia is so severe that renal water excretion is overwhelmed, or in conjunction with SIADH, reset osmostat, or drug effects, all of which may be associated with psychosis.

Clearly, further work is required to unravel the basis of these troubling phenomena which may then lead to more effective ways of managing polydipsia and water intoxication.

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The Psychiatric Symptoms, Diagnoses and Care Needs of 100 Mentally Handicapped Patients

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One hundred randomly selected residents of a mental handicap hospital originating from Dundee were interviewed using a standardised assessment based on the modified Standardised Clinical Interview Schedule. Information on certain behavioural items and self-care skills was obtained from nursing staff and case records. An ICD–9 diagnosis could be made for 80 individuals, including diagnoses usually reserved for children. Thirty subjects were regarded as needing long-term psychiatric mental handicap hospital care. All but one of the remaining 70 subjects required a staffed residential placement and all but 15 some form of out-patient or short-term in-patient provision from specialist health services. The findings indicate a need for approximately 30 psychiatric mental handicap beds per 100 000 population.

In the past, many mentally handicapped patients who would not now be considered to require hospital care were placed in hospital for lack of more suitable alternatives. In recent years, smaller, locally based and domestic-type facilities have been advocated as offering a more normal and acceptable lifestyle (Scottish Health Services Planning Council, 1988). As a result of this change in policy, there has been a substantial reduction in the number of residents in mental handicap hospitals over the last decade. However, various studies have shown a high prevalence of psychiatric morbidity in mentally handicapped people (Reid, 1982; Gostason, 1985; Glue *et al*, 1988). Some of this morbidity corresponds to traditional psychiatric illness, but much of it defies definition. It is important to look critically at this area since severe psychopathology and behaviour disorder are among the most influential factors leading to hospital placement.

The aim of the present study was twofold:

(a) to define the psychiatric symptoms of a random selection of mentally handicapped adult in-patients, not just in terms of symptoms and overall severity, but also as far as possible in terms of standardised psychiatric classification using ICD–9 (World Health Organization, 1978)

(b) to identify patients' psychiatric and residential care needs, in relation both to the psychiatric disorder and to other characteristics.

Method

Strathmartine Hospital serves the needs of the mentally handicapped in Tayside (population approximately 400 000). The number of patients in the hospital has fallen sharply, from 586 in 1976 to 347 in 1988. Of 168 patients from the city of Dundee, 100 were selected for the present survey using a random-number table. Individuals under the age of 16 years were excluded.

For each patient we noted the following characteristics: age, sex, cause (if known) of mental handicap, degree of retardation, whether or not there was significant impairment of hearing, vision or mobility, and whether the patient was epileptic. We adopted the same definition of epilepsy as in our previous studies (Reid *et al*, 1978). Information about self-care was also collected, under standard headings including walking, continence, dressing, feeding skills and communication.

Each patient was assessed as described by Reid *et al* (1978). Nursing staff who knew the patient well (usually the charge nurse) were asked whether the patient had shown significant abnormalities in respect of the following behavioural problems over the preceding week: withdrawal; overactivity; stereotypies; irritability, violence or aggression; noisiness; self-injury; stripping; feeding disturbance; stealing; inappropriate sexual behaviour; inappropriately immature behaviour. These were rated on a scale of 0–4. A note was also made of recordings of these symptoms made in the case notes in the previous five years under the same headings.

The patient was interviewed for not less than 20 minutes by one of three consultant psychiatrists (BRB, CBB, AHR) using the modified version of the Standardised Clinical Interview Schedule, which we have shown to be reliable and valid in mentally handicapped adults (Ballinger *et al*, 1975). In patients with poor verbal ability only the section on manifest abnormality was completed, but in all cases an overall rating was made on a scale of 0–4.

A diagnosis was made according to ICD–9 where a psychiatric condition was present and could be identified.

Health care needs were identified under standard headings including: long-term psychiatric in-patient care; short-term psychiatric in-patient care; medical psychiatric out-patient care; non-medical psychiatric out-patient care; other residential care within the National Health Service

(NHS); other NHS non-residential care. The use of the word 'psychiatric' implied specialised psychiatric services for the mentally handicapped.

Residential care needs were defined under the following headings: (a) psychiatric in-patient care – for patients with severe behaviour disorder or psychiatric illness requiring 24-hour provision of psychiatric nursing and medical attention; (b) nursing home – for patients with a degree of mental or physical disorder requiring continuous nursing care but not of the specialised nature provided in a psychiatric hospital; (c) augmented hostel – for patients not requiring nursing attention but needing more supervision and training than at present provided in ordinary hostels because of lack of personal skills or mild psychiatric or behaviour disorder in the patient; (d) ordinary hostel – for patients requiring general supervision and care with 24-hour staff presence (this would include residential establishments staffed to the present basic allowance from the Department of Social Security, which finances approximately 4.5 staff for a 10-place community house where residents would be expected to have basic dressing and hygiene skills and minimal domestic skills without significant behaviour disturbance); (e) residential care for the elderly – for elderly patients with a degree of physical and mental dependence usually accepted in mainstream residential care for the elderly; (f) group home – for small groups of patients not requiring 24-hour staff supervision but with access to support staff or members of the community mental handicap team, or both.

Results

One hundred patients (55 men, 45 women) were assessed (age range 19–84 years, mean 43.2, s.d. 7.21). The duration of stay varied from 1 to 77 years with a mean of 24.4 years. The degree of handicap in terms of ICD–9 classification was: borderline (2), mild (19), moderate (26), severe (39), and profound (14). In 49 cases the likely or possible cause of mental handicap was known, including 10 instances of Down's syndrome, 18 of birth injury or cerebral palsy, and 11 cases of infection.

Sixty individuals were currently receiving psychiatric drugs including: neuroleptics (35 patients), antidepressants (4), anxiolytics (9), lithium (7), and anticonvulsants (24). Some patients were on more than one agent. Self-care skills were categorised as follows: able to walk unaided (84); fully continent (53); able to dress unaided (38); effective speech (71); ability to feed without help (64). Twenty-eight patients were epileptic. Fourteen had partial or complete blindness, and eight were partially or completely deaf.

The various symptoms were recorded in the following numbers of individuals: withdrawal (34); overactivity (24); stereotypies (43); irritability, violence or aggressiveness (62); noisiness (35); self-injury (25); stripping (12); feeding disturbance (25); stealing (7); inappropriate sexual behaviour (21); inappropriately immature behaviour (34); and other abnormalities (45).

Fifty-nine patients had one or more manifest abnormalities of behaviour rated as pathological (2–4) on the modified Standardised Interview (Table 1). Thirty patients were noted to avoid eye contact to a pathological degree. Forty-one patients had no individual ratings in the 2–4 range. The overall ratings on the modified Standardised Interview were as follows: 0 (9); 1 (27); 2 (34); 3 (21); 4 (9).

Table 1

Manifest abnormalities from the Standardised Clinical Interview Schedule and need for long-term in-patient mental handicap psychiatric care among 100 mentally handicapped people

	No. scoring 2–4 for abnormality	No. needing in-patient care
Slow	10	4
Suspicious	2	1
Histrionic	9	3
Depressed	1	1
Anxious	6	2
Elated	3	2
Flattened affect	17	9
Delusions	1	0
Hallucinations	3	1
Concern with bodily function	1	1
Depressive thoughts	0	0
Overactivity	11	6
Distractibility	14	4
Stereotypy	40	14
Irritability	9	6
Lability	0	0
Pica	1	0
Self-injury	6	2

It was possible to make at least one psychiatric diagnosis for 80 individuals, although sometimes this involved using categories mainly intended for diseases of childhood. The main categories diagnosed are shown in Table 2.

The health care needs were as follows: none (15); short-term in-patient psychiatric care (10); medical psychiatric out-patient care (24); non-medical psychiatric out-patient (e.g. community nurse or clinical psychologist) (41); other NHS out-patients (20); continued care in hospital (30). Some individuals required more than one category of service provision.

Table 2

ICD–9 psychiatric diagnosis and long-term hospital care needs of 100 mentally handicapped people

	No. with main psychiatric diagnosis	No. needing in-patient care
Organic psychosis (290,294)	3	0
Schizophrenia (295)	5	1
Affective psychosis (296)	5	2
Paranoid states (297)	3	0
Psychosis of childhood (299)	12	8
Personality disorder (301)	17	8
Sexual deviation and disorders (302)	4	2
Special symptoms or syndrome (307)	5	0
Non-psychotic mental disorder following brain damage	2	0
Disturbance of conduct (312)	15	0
Hyperkinetic syndrome of childhood (314)	9	7
None	20	2

Table 3
Need for long-term in-patient psychiatric mental handicap care and degree of handicap

	No. of patients with degree of handicap	No. needing psychiatric long-term care
Borderline and mild	21	3 (14%)
Moderate	26	7 (27%)
Severe	39	15 (38%)
Profound	14	5 (36%)
Total	100	30

Residential care needs were determined as follows: long-term psychiatric in-patient (30); nursing home (29); augmented hostel (29); ordinary hostel (8); group home (1); residential home for the elderly (3).

Table 3 shows the relationship between degree of handicap and the need for continued long-term in-patient hospital care. This indicates a trend for a higher proportion of those with more severe mental handicap to require in-patient care, but this was not statistically significant. None of the ten Down's syndrome patients were thought to require hospital care, whereas 5 of the 11 patients with handicap following infection were judged to require long-term hospital facilities. The 53 patients with severe or profound handicap included 26 of those 30 individuals showing an overall psychiatric illness rating on the Standardised Interview in the more severe categories of 3 and 4.

Table 4 shows the overall ratings for psychiatric disturbance in relation to the need for long-term in-patient psychiatric care. Patients with the more severe disturbance (3–4) were significantly more likely to require in-patient care ($\chi^2=41.3$, d.f. $=1$, $P<0.01$).

The rated manifest abnormalities and need for in-patient psychiatric care are shown in Table 1. Although the numbers on several of the ratings are small, it can be seen that no individual symptoms were judged to make in-patient care mandatory. Similarly, the staff ratings of self-care were not reliable predictors of the need for continued in-patient care.

The health care needs of patients with various psychiatric diagnoses are shown in Table 2. Only 3 of the 13 patients with functional psychoses were thought to require long-term in-patient care, in contrast to 8 of the 12 individuals with childhood psychoses (although this does not achieve statistical significance: $\chi^2=3.20$, d.f. $=1$). Eight of the 17 patients with personality disorders were thought to require continued in-patient hospital care, in contrast to none of the 15 patients with conduct disturbance (Fisher's exact test, $P<0.01$).

Among those patients judged to require long-term in-patient hospital care, 18 were men (33% of the male population) and 12 were women (27% of the female population).

Table 4
Need for long-term in-patient psychiatric mental handicap care and overall rating for psychiatric disturbance

	No. with overall rating of psychiatric disturbance	No. of patients needing long-term hospital care
0	9	0
1	27	0
2	34	7
3	21	15
4	9	8

Discussion

This survey confirms previous reports both from the Dundee Mental Handicap Service, and elsewhere, that there is a high proportion of psychiatric disturbance in the in-patient population of mental handicap hospitals (Reid, 1982). The overall figure of 64% scoring 2–4 on the modified Standardised Interview is higher than the figures obtained for the same population in previous studies. For example, in 1977, 31% were rated in this range for psychiatric disorder (Ballinger & Reid, 1977). During this period the population of the hospital has been nearly halved and this suggests that there is now a greater concentration of more severely psychiatrically ill patients. Associated with this is a widespread use of psychotropic drugs (in 60% of patients if anti-convulsants are included).

There has been debate about the applicability of the standardised psychiatric diagnostic criteria of ICD–9 in this population (Russell, 1988). We have previously reported on the diagnosis of functional and organic psychotic disorders (Reid, 1982) and personality disorder in this population (Reid & Ballinger, 1987). In the present study we were able to make an ICD–9 diagnosis in 80 individuals, although not all currently suffered from severe symptoms, and this is similar to the 71% figure quoted by Glue *et al* (1988). However, this use of ICD–9 sometimes involved the use of diagnostic groupings normally related to child psychiatry, where these seemed more appropriate. Inevitably the figures rest on the clinical judgement of the raters, and should be subjected to further assessment of reliability in the future. We did, however, feel it was possible to apply the usual psychiatric criteria to many, if not all, individuals, and this may be preferable to devising a completely separate diagnostic system (Lund, 1985). It would be useful and interesting to repeat this exercise using DSM–III–R and ICD–10.

The judgements of the health and residential care needs are inevitably based on the opinions of the raters and are thus to some degree subjective. Only 30% were regarded as requiring long-term care in a mental handicap hospital but 85% were thought to have specialist health care needs extending beyond primary care, including out-patient provision from specialist medical and other staff. This finding emphasises the continuing need for specialist handicap input from medical, nursing, clinical psychology and other paramedical staff, but organised on a day-patient, out-patient and domiciliary basis rather than centred on in-patient care. The transfer of patients to community residential placements does not indicate a reduction in need for these specialist skills but a reorganisation and development to provide the necessary support, treatment and training for mentally handicapped individuals in the community.

All but one of the subjects were thought to require some form of residential placement, but in 69 this was judged not to be a mental handicap hospital, although an element of nursing would be required for all those individuals in nursing homes. Most of the hostel and nursing-home requirements described would need to be specially committed to the care needs of the mentally handicapped. Inevitably these judgements make some assumptions about the nature of the units concerned and those establishments taking more handicapped or disturbed individuals will require an input from specialist professional staff to advise on management.

As expected, most of those with more severe psychiatric disorders were thought to require long-term hospital placement. However, hospital placement was not considered necessary for some patients who were rated highly on psychiatric illness if they were not disruptive. There was a tendency for the diagnoses of personality disorder and childhood psychosis to be more associated with a perceived need for hospital care than the diagnosis of functional psychosis and conduct disturbance. This emphasises the significance of personality problems in mentally handicapped people (Zigler & Burack, 1989).

Finally, although psychiatric and behavioural symptoms were very widespread in this population, no individual symptom implied an inevitable need for long-term hospital care.

We believe it is right that only those for whom it is essential should be kept in long-term psychiatric-type mental handicap hospital care. Others will, however, require a wide range of residential and community provision geared to their needs, including short-term psychiatric admission and extensive community specialist services. If these findings are extrapolated to the whole hospital population they would point to the need for just over 100 hospital beds for long-term care of the adult mentally handicapped population in Tayside. Taking into account shorter-term and respite care, a total bed requirement of approximately 120 would be necessary (30 per 100 000 population), and this is similar to the figure quoted by Day (1986). However, until appropriate residential provision with adequately trained and skilled staff accompanied by necessary day-care places and professional support are available in the community, it will not be possible to reduce hospital provision to this level.

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The Factor Structure and Factor Stability of the Hospital Anxiety and Depression Scale in Patients with Cancer

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An exploratory factor analysis of the HAD was carried out in 568 cancer patients. Two distinct, but correlated, factors emerged which corresponded to the questionnaire's anxiety and depression subscales. The factor structure proved stable when subsamples of the total sample were investigated. The internal consistency of the two subscales was also high. These results provide support for the use of the separate subscales of the HAD in studies of emotional disturbance in cancer patients.

With the growth of liaison psychiatry as a specialty in its own right, the need for standardised measures of emotional disturbance in patients with physical illness has become increasingly important. The most commonly encountered emotional disorders in medical patients are anxiety and depression, which frequently coexist. Scales for investigating both these symptoms are therefore particularly attractive to researchers and clinicians in this field. One such scale which is receiving attention is the Hospital Anxiety and Depression Scale (HAD). This is a brief, self-administered rating scale which has been specifically designed for patients with physical illness (Zigmond & Snaith, 1983). It consists of 14 items, seven regarding depression, seven anxiety. The depression subscale has been constructed so that somatic items are largely excluded. The subscale heavily emphasises anhedonia, which is considered by some to be the symptom of depression characteristic of the endogenous subtype and predictive of response to antidepressant medication (Klein, 1974). The items on the anxiety subscale were chosen by Snaith *et al* (1982) from anxiety items in the Present State Examination (Wing *et al*, 1974) and from Snaith's own research.

Evidence for the concurrent validity of the HAD has been reported in psychiatric patients (Bramley *et al*, 1988), in a heterogeneous group of patients with physical illness (Aylard *et al*, 1987) and in patients attending a genitourinary clinic (Batczak, 1988). Ibbotson *et al* (1989) investigated the validity of the HAD as a screening instrument for psychological distress in 514 patients with cancer. The HAD was compared with two other self-rating scales, the General Health Questionnaire (GHQ; Goldberg, 1972) and the Rotterdam Symptom Checklist (RSCL; de Haes *et al*, 1983). All three scales were also assessed by comparison with the Psychiatric Assessment Schedule (PAS; Maguire *et al*, 1978) which was used as a gold standard: the PAS is a semistructured interview that includes symptoms based on DSM–III criteria (American Psychiatric Association, 1980). Ibbotson *et al* concluded that the HAD performed best overall, but the RSCL performed better as a screening instrument for patients with stable disease.

These validation studies assume that the HAD does, as its authors suggest, function as two scales measuring two distinct mood states. Clinical experience with patients with physical illness, however, suggests that it is frequently difficult to distinguish anxiety and depression. In view of this it could be argued that the HAD would be more validly used as a single 14-item measure of mixed emotional disturbance: it might act as a global measure of psychological distress, as does the GHQ. Lewis & Wessely (1990) have used the HAD in this way with dermatology patients, while Razavi *et al* (1990) used the full 14-item scale in a study of Belgian cancer patients. On the basis of a factor analysis of the data, Razavi *et al* concluded that the HAD was a unidimensional measure of emotional distress.

It is of both theoretical and practical importance to establish whether the HAD should be used as a unidimensional or bidimensional scale. This paper describes the factor structure of the HAD in 568 cancer patients, and is the first detailed analysis of the scale to be reported with any substantial group of patients. The use of factor analysis as a method of assessing the validity of an empirical measure is well established (Nunnally, 1978). In this study it has been used to test for the existence of two underlying factors corresponding to the subscales of the HAD.

Method

The HAD is currently being used as a screening instrument in a larger study by the Psychological Medicine Group at the Royal Marsden Hospital. The plan of investigation has been described in detail elsewhere (Moorey & Greer, 1989). In brief, a consecutive series of cancer patients attending the Royal Marsden Hospital is screened to select patients with psychological morbidity. These patients are then invited to join a randomised controlled trial comparing a brief psychological therapy with a no-treatment control. Since the HAD is one of the screening instruments, anxiety and depression scores are available from a large sample of cancer patients with a wide range of psychological distress, from none to pathological. To enter the study and receive the HAD as part of the screening procedure patients need to fulfil the following criteria:

(a) patients attend the Royal Marsden Hospital with a diagnosis of any form of cancer except cerebral tumours at initial diagnosis of cancer or first recurrence
(b) patients are aware of their diagnosis
(c) estimated duration of survival, as judged by the clinician, is more than 12 months
(d) patients are aged 18–75 years
(e) patients speak fluent English
(f) patients have no obvious intellectual impairment
(g) patients reside within the Greater London or Home Counties area.

Patients who meet the entry criteria are seen between 4 and 12 weeks after initial diagnosis or first recurrence. This time lag allows patients to recover from the initial shock of learning of their diagnosis. Patients screened earlier would be more likely to be experiencing transient disturbances of mood. Although these patients may vary in the stage and severity of their cancer, they all share the same characteristic of being within three months of learning that they have cancer, or a recurrence of cancer.

The 575 patients who had been screened during the first 18 months of the study were included in this investigation.

Factor analyses were performed on the full 14-item HAD scale, and on the two seven-item subscales separately. Using the BioMedical Data Package (BMDP, 1988), Pearson product-moment correlation coefficients were computed and a principal-components procedure used to extract the initial factors. The predetermined criterion chosen for the number of factors to be extracted was the commonly used Kaiser or eigenvalue criterion, with which factors are retained if they have an eigenvalue equal to or greater than 1.

Two methods of rotation of this factor solution were compared: orthogonal rotation using the varimax procedure, and oblique rotation using direct quartimin (the oblique rotational procedure recommended for use with BMDP). Following the initial extraction of factors, rotation is used to achieve the simplest and most meaningful factor structure. An orthogonal rotation makes the assumption that the underlying factors are not correlated. An oblique rotation, however, does not make this assumption, and so allows for the possibility that the factors might show a correlation with each other. There is considerable debate over whether anxiety and depression exist as separate entities (Stavrakaki & Vargo, 1986) and subscales of anxiety and depression on questionnaires frequently show high correlations. For this reason it was considered important to compare the two rotation procedures.

In order to test the stability of the factor structure obtained, further analyses of two subsamples were carried out. (a) The sample was split into two halves, and factor analyses were then performed on these two subsamples. (b) Separate factor analyses were performed on the data from male and female subjects to establish the factor stability across sexes.

Results

Results from the screening questionnaires were available for 575 patients. The mean age of the sample (70.8% men, 29.2% women) was 55.1 (s.d. 12.8) years. A wide range of cancer diagnoses was covered. The largest diagnostic category was breast cancer (47.1%), while 9.7% of patients had malignancies of the cervix, uterus or ovary, 8.7% non-hodgkin's lymphoma, 7.3% head and neck tumours, and 5.7% Hodgkin's disease. Other diagnoses constituted 21.5% of the sample. The sample was heavily weighted towards those experiencing a first episode of cancer: 92.5% of patients had primary disease while only 7.5% had recurrent disease. This bias was reflected in the stage of disease recorded: 62.7% had local disease, 26.9% loco-regional disease, and 10.4% metastatic disease. A further biasing factor was the exclusion of any patients from the study who had a life expectancy of less than one year. The World Health Organization's (WHO, 1979) performance status criteria were used to record the degree to which patients were disabled by the disease: 59% of the patients were able to carry out all normal activities, 33% experienced some difficulty with strenuous activity, 6% were unable to work, but able to carry out all self-care, and only 1% were limited in their self-care or restricted to bed for more than 50% of the time.

The mean scores on the HAD were 5.44 for anxiety (s.d. 4.07; range 0–19) and 3.02 for depression (s.d. 2.98; range 0–15). Zigmond & Snaith (1983) recommend that scores of 8 or more on a subscale are taken to indicate possible pathology. Using this cut-off point, 27% of patients were in the range for clinical anxiety, and 8.7% for depression. Individual HAD items (possible scores of 0, 1, 2, 3) varied between a minimum of 0.23 (item 11) and 1.23 (item 8).

Complete sets of HAD scores were available for 568 patients, and factor analyses were carried out on these. Two factors emerged which accounted for 53% of the variance. An orthogonal rotation was performed; using the criterion of loading of 0.45 as a cut-off point, the anxiety and depression items loaded onto separate factors with the exception of item 7, from the anxiety subscale – "I can sit at ease and feel relaxed."

An oblique rotation was then performed (Table 1). Again all the items except item 7 loaded on the appropriate factor. It is to be expected that levels of anxiety and depression will not be entirely independent in this population, and this is supported by the finding of a correlation of 0.50 between the two factors when an oblique rotation is performed. It was considered that oblique rotation provides the most psychologically meaningful way of analysing the data in this sample. This method of rotation was used in the rest of the study to test the stability of the factor structure.

Table 1
Factor loadings of HAD items (oblique rotation)

HAD item	Factor 1 (depression)	Factor 2 (anxiety)
Anxiety subscale		
(1) I feel tense or wound up	0.13	0.71
(3) I get a sort of frightened feeling as if something awful is about to happen	0.09	0.77
(5) Worrying thoughts go through my mind	0.17	0.72
(7) I can sit at ease and feel relaxed	0.51	0.30
(9) I get a sort of frightened feeling like 'butterflies' in the stomach	−0.10	0.86
(11) I feel restless as if I have to be on the move	−0.07	0.66
(13) I get sudden feelings of panic	−0.01	0.83
Depression subscale		
(2) I still enjoy the things I used to enjoy	0.78	−0.02
(4) I can laugh and see the funny side of things	0.74	−0.06
(6) I feel cheerful	0.76	−0.01
(8) I feel as if I am slowed down	0.51	0.14
(10) I have lost interest in my appearance	0.49	0.07
(12) I look forward with enjoyment to things	0.79	0.01
(14) I can enjoy a good book or TV programme	0.65	−0.08

Principal-components analysis was carried out on the two subscales of the HAD separately. Analysis of the HAD anxiety scale resulted in a single factor which accounted for 57% of the variance. Analysis of the HAD depression scale revealed a single factor which accounted for 47% of the variance. Two reliability estimates for the subscales were calculated. Carmine's theta for the anxiety scale was 0.87 and for the depression scale 0.81. Cronbach's α was 0.93 for the anxiety scale and 0.90 for the depression scale.

Table 2
Factor loadings of split halves of sample (oblique rotation)

HAD item	Depression factor		Anxiety factor	
	sample 1	sample 2	sample 1	sample 2
Anxiety subscale				
1	0.15	0.13	0.71	0.70
3	0.17	0.01	0.69	0.86
5	0.19	0.16	0.72	0 72
7	0.51	0.50	0.28	0.36
9	−0.04	−0.15	0.80	0.90
11	−0.14	0.03	0.66	0.65
13	0.00	−0.02	0.82	0.83
Depression subscale				
2	0.74	0.81	0.12	−0.12
4	0.71	0.76	−0.05	−0.05
6	0.68	0.80	0.05	−0.02
8	0.51	0.47	0.16	0.16
10	0.59	0.35	−0.03	0.22
12	0.76	0.81	0.06	0.00
14	0.65	0.61	−0.16	0.04

Table 3
Factor loadings for men and women (oblique rotation)

HAD item	Depression factor		Anxiety factor	
	men	women	men	women
Anxiety subscale				
1	0.18	0.14	0.68	0.70
3	0.23	0.05	0.62	0.81
5	0.25	0 17	0.70	0.70
7	0.59	0.48	0.23	0.33
9	−0.12	−0.07	0.82	0.85
11	−0.05	−0.11	0.62	0.72
13	−0.11	0.06	0.82	0 80
Depression subscale				
2	0.82	0.75	−0.07	0.01
4	0.80	0.71	−0.12	−0.03
6	0.66	0.82	0.12	−0.09
8	0.61	0.42	0.11	0.14
10	0.61	0.42	0.02	0.11
12	0.83	0.79	0.05	−0.02
14	0.61	0.64	−0.09	−0.05

To test the stability of the factor structure obtained, the sample was split into two halves by taking the first 285 cases and comparing them with the last 283 cases. There were no significant differences between these two subsamples in terms of age, sex, or performance status. There were, however, significantly more patients with locoregional disease (as opposed to local or metastatic disease) in the second sample ($\chi^2 = 12.50$, 2 d.f., $P = 0.002$). A principal-components analysis was computed on each sample and oblique rotation performed. Table 2 shows the factor loadings for the two samples. For sample 1 two factors emerged explaining 52% of the variance, while for sample 2 two factors also emerged which explained 54% of the variance. The factor structure of the two samples is the same, and identical to the factor structure of the full sample of 568 cases.

Principal-components analysis followed by oblique rotation using direct quartimin in 167 men and 401 women revealed two factors in both groups. A correlation of 0.37 was found between the two factors in men. In women this correlation was 0.55. For the majority of items the factor loadings were very similar (Table 3). In women two items from the depression subscale just failed to load on the depression factor, item 8 "I feel as if I am slowed down" and item 10 "I have lost interest in my appearance".

Discussion

These results support the view that in the group of cancer patients studied, the HAD is bidimensional, tapping the separate but related constructs of anxiety and depression. Principal-components analysis consistently extracted two factors, in both the full sample of 568 patients and in the subsamples. The HAD is a relatively new instrument, and only one previous study has examined its factor structure in cancer patients (Razavi *et al*, 1990).

Razavi *et al* interviewed 226 Belgian cancer inpatients using the Diagnostic Interview Schedule (DIS; Spitzer, 1983) and validated the Belgian translation of the HAD against this measure. They briefly report that a preliminary factor analysis failed to show the bidimensionality of the scale in the sample tested, and so used the total HADS score as a psychological distress scale. There is not sufficient description of the statistical tests used to make a full comparison of the two studies. If the difference is not a product of differing statistical techniques, it is likely to be due to some difference in the nature of the populations studied. This may be a reflection of cultural or translation differences, or the composition of the two samples of cancer patients might affect the way the instrument performs. Although the Belgian sample is comparable in the types of cancer represented, age, range and sex distribution, all the patients were in hospital, whereas in the present study most were out-patients. While breast cancer represented the largest group in the Belgian study as in our own, other types of cancer were represented in larger numbers. Razavi's patients had more advanced disease and were more disabled by the disease than the group reported here.

One final possible difference concerns the HAD scores in these two populations. Unfortunately, Razavi does not report the separate anxiety and depression scores in his patients. Our cancer patients displayed substantially more anxiety than depression (27% in the clinical range for anxiety and 8.7% in the clinical range for depression). Patients with more advanced disease might be expected to experience more hopelessness and depression. It is not clear to what extent and in what way the factor analysis of our sample may have been affected by this preponderance of anxiety over depression. In the light of the differences between the present study and that of Razavi *et al*, our findings of the psychometric properties of the HAD can only be applied to patients with early-stage cancer. The instrument may not display the same properties when used with those with more advanced disease.

Our results suggest that the items on the two subscales of anxiety and depression discriminate very well. This applies to both orthogonal and oblique rotations. The only item which does not perform well is item 7 ("I can sit at ease and feel relaxed"). This item is from the anxiety subscale but actually loads on both factors. This difference applies more to women than men, but there appears to be no clear explanation for why this item should not be correlated more highly with anxiety. Further studies are needed to establish whether this is an enduring finding.

Although the two factors extracted correspond well to the two subscales, these factors only contribute 53% of the variance, leaving nearly half the variance unexplained. This result, however, compares well with other factor-analytic studies of questionnaires of this kind. Gould (1982) identified five factors which explained 58.7% of the variance in the 21-item Beck Depression Inventory (BDI; Beck *et al*, 1961). O'Brien & Glaudin (1988) examined the factor structure of the Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960) and found that the factors accounted for only 40% of the variance. In a recent study using the GHQ in a sample of 6000 subjects in the community (Huppert *et al*, 1989), six factors were identified which accounted for 50.6% of the variance. It is of some interest that these questionnaires were consistently found to produce five or six factors, even though the BDI and HRSD are measures of a single mood state – depression. Authors have taken these findings to be evidence for the multidimensional nature of the depressive syndrome.

Factor analysis of the HAD, by contrast, extracted two factors. Factor analysis of the separate subscales for anxiety and depression produced unidimensional solutions for both, although this is perhaps not surprising given the small number of items in each subscale. Further evidence for the homogeneity of the two subscales of the HAD are their high-coefficient α values (anxiety, 0.93; depression, 0.90). For a scale to be used for research purposes, Nunnally (1978) recommends that coefficient α is at least 0.6, while for it to be used as a screening instrument it should be at least 0.8. Using these criteria both the HAD subscales can be justifiably used as screening measures. This homogeneity is a distinct advantage for the HAD, but it may also be a weakness. Significant aspects of the syndromes of anxiety and depression are omitted. This does not just apply to somatic symptoms which have been deliberately excluded. Important components of depression, for instance, such as hopelessness, guilt and low self-esteem are not assessed, because the scale measures only features of anhedonia. This is perhaps not so important in screening studies as it is in outcome studies.

At the moment the HAD seems to be the best instrument available for simple and rapid evaluation of psychological interventions in patients with physical illness. It is well established that the component symptoms of the syndromes of anxiety and depression do not all respond at the same pace to treatment. In this case more comprehensive scales might prove more sensitive to change. Studies of how the HAD changes in response to treatment in comparison with other scales are required.

In addition to its high internal consistency, the questionnaire has a reliable factor structure. The same two factors emerged from analyses of two halves of the sample, and from separate analyses of male and female subjects. In this large sample of cancer patients the factor structure is robust.

These results confirm that the HAD is a useful instrument for measuring anxiety and depression in cancer patients, and that for patients with early cancer the continued use of its two subscales is justified. Further studies are needed to establish whether this also applies to patients with advanced cancer and other types of physical illness.

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Gender-Divergent Aetiological Factors in Obsessive–Compulsive Disorder

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Among 307 adults with OCD, early onset (age 5–15 years) was more common in men and later onset (age 26–35 years) in women. Early onset was associated with more checking, and late onset with more washing. More women than men had a history of treated depression; 12% of the women but none of the men had a history of anorexia. More women than men were married. Gender-divergent features may reflect differential aetiological factors. Our sample resembled others in the literature in its slight overall female preponderance, low rate of marriage and low fertility, onset mainly before age 35 years, chronicity, and common present and past depression.

Obsessive–compulsive disorder (OCD) was the fourth most common psychiatric disorder in the US population in the National Epidemiologic Catchment Area (ECA) program (Robins *et al*, 1984; Karno *et al*, 1988). Many have reported its demographic and clinical features (Pollit, 1957; Ingram, 1961; Kringlen, 1965; Lo, 1967; Welner *et al*, 1976; Coryell, 1981; Rasmussen & Tsuang, 1986; Steketee *et al*, 1987), but few have noted the differential effect of gender on some aspects of OCD and the potential aetiological relevance of this. We report on this issue in 307 patients who met DSM–III criteria (American Psychiatric Association, 1980) for OCD.

Method

Our total of 307 patients with OCD came from two samples. In the first, prospective sample (group 1), 172 patients, referred consecutively to a treatment trial for rituals at the Maudsley behavioural unit between 1980 and 1983, were screened. A total of 145 of these (68 men, 77 women) met DSM–III criteria for OCD at a semistructured interview carried out by two psychiatrists, who recorded demographic data, obsessive–compulsive (OC) symptoms, depression (Research Diagnostic Criteria (RDC); Spitzer *et al*, 1978), and past and family history of treated psychiatric disorder.

In the second, retrospective sample (group 2), two psychiatrists examined the case notes of all 222 patients with a primary diagnosis of OCD (ICD–9 300.3; World Health Organization, 1975), who had received behavioural treatment between 1977 and 1980 in the same unit (90% outpatients). Of these, 60 had inadequate data. The remaining 162 patients (69 men, 93 women) met DSM–III criteria for OCD. The demographic data (sex, age, age of onset, illness duration and marital status) were similar to that of group 1 and were pooled. The phenomenology of OC symptoms and the data concerning psychiatric history were incomplete and so will not be presented.

Table 1
Gender comparison of characteristics of total sample ($n = 307$ unless otherwise stated)

Characteristic	Men ($n = 137$)	Women ($n = 170$)
Mean (s.d.) age: years	35 (10.6)	34 (11.2)
Mean (s.d.) age of onset: years	21 (9.3)	24 (9.2)*
Mean (s.d.) duration of illness: years	14 (10.1)	10 (9.4)*
% with duration of illness of:		
<5 years	22	45*
>20 years	25	13*
% with marital status of:		
single	54	32**
married	39	60
divorced	7	5
No. (%) with birth order of:[1]		
first born[2]	30 (49)	34 (44)
later born	31 (51)	43 (56)
only child	13 (19)	9 (11)
Mean no. of:[3]		
siblings	2 9	3.1
children	1 1	1 7
% with rituals of:[3]		
cleaning	57	75
checking	70	55
ruminations	63	47
% with past history of:[3]		
depression	32	51*
alcohol abuse	8	0*
anorexia	0	12***

*$P<0$ 01, **$P<0$ 02, ***$P<0$ 001.
1. $n = 138$.
2. Includes 'only children'.
3 $n = 145$ (from group 1)

In both groups patients had obsessions and/or compulsions that were not due to another mental disorder and were severe enough to interfere with social or personal functioning. A minority of cases however did not regard their obsessions as senseless, and a larger number did not resist them.

Significant differences refer to t-tests or χ^2 as appropriate.

Results

Demographic features

A gender comparison of the demographic features of the total sample is shown in Table 1. Of the total, 45% ($n = 137$) were men and 55% ($n = 170$) were women ($P < 0.02$). The mean age of presentation of 34.5 years (range 16–67 years) was similar in both sexes. By contrast, mean age of onset of OCD was slightly but significantly earlier in men than in women (21 v. 24 years; $P < 0.01$). More men than women had an early onset of OCD, from age 5 to 15 years ($P < 0.01$), and over twice as many women than men had a later onset, from age 26 to 35 years ($P < 0.01$); at the peak age of onset for both sexes (16–25 years) the male:female ratio was 1:1 (Table 2). In keeping with this, men had a longer duration of illness than did women (14 v. 10 years; $P < 0.01$) (Table 1). Twice as many men than women had a problem duration of over 20 years ($P < 0.01$) and more women than men had a duration of less than five years ($P < 0.01$).

The number married was low for a sample with a mean age of 34 years: 42% overall were single (54% men, 32% women; $P < 0.02$) (Table 1). Fertility, too, was low: 30% of marriages were childless. Duration of marriage was not recorded. Fifteen per cent were only children (men 19%, women 11%); 46% of cases were first born (men 49%, women 44%), including only children.

Other clinical features (group 1)

Of group 1, 93% of cases had obsessions and rituals, 2% had only obsessions and 5% had obsessions and avoidance but no rituals. A total of 57% of men compared with 75% of women had cleaning rituals ($P < 0.1$), and 70% of men compared with 55% of women had checking rituals ($P < 0.1$). Compared with late-onset cases, early-onset patients exhibited more checking rituals (38% v. 10% $P < 0.001$) and fewer washing ones (17% v. 45% $P < 0.01$). The two groups did not differ in the proportion of patients who had both checking and washing rituals.

In a subgroup of this sample ($n = 49$) who had been treated in a research trial, the early-onset v. late-onset patients did not differ significantly in severity, social adjustment or response to treatment.

Only 18% of our group 1 cases were seeing a psychiatrist for the first time, 75% having been previously treated for OCD. Many had also had past treatment for other problems, especially for depression. More women (51%) than men (32%) had a history of treated depression ($P < 0.01$). Patients with a later age of onset of OCD were more likely to be treated for depression. At the time of presentation, 14% were diagnosed as having secondary major depression and 11% as having dysthymic disorder according to RDC.

Table 2
Percentage of males and females with onset of OCD in various age ranges ($n = 305$)[1])

Onset age: years	Men (%)	Women (%)
5–15	35	20*
16–25	43	41
26–35	13	32*
36+	9	7

*$P < 0.01$.

1 Age of onset not available in two cases.

Other problems for which group 1 cases had been treated were alcohol dependence (8%, more often in men – $P < 0.01$) and anorexia (12% of the women, none of the men – $P < 0.001$). Features of those with a history of anorexia (all women) have been reported elsewhere (Kasvikis *et al*, 1986); compared with non-anorexic women with OCD, they were younger, more often single, and developed OCD earlier, but they had similar types of rituals.

Discussion

A hitherto neglected finding in both our samples was that more men had an onset of OCD during childhood (aged 5–15 years) and far more women had a later onset (aged 26–35 years). Only one previous study included similar data; in a Swedish sample of 251 subjects with OCD, significantly more men had an age of onset of under 20 years, while the age of onset for women tended to be between 25 and 40 years (Skoog, 1965). The same effect of gender on age of onset was found in 32 US patients (14 men, 18 women) with OCD (DSM-III criteria) (data kindly supplied by Professor Edna Foa); 64% of her male compared with 27% of her female patients had an age of onset between three and 15 years, while 7% of her male compared with 38% of her female patients had an age of onset of 26–35 years. Further evidence of early onset being more common in males is seen in eight studies of OCD in children and adolescents (Berman, 1947; Despert, 1955; Judd, 1965; Adams, 1973; Hollingworth *et al*, 1980; Bolton *et al*, 1983; Apter & Tyano, 1988; Swedo & Rapoport, 1989). They reported a total of 174 boys and 70 girls with OCD, a ratio of 5:2.

The aetiology of OCD is likely to involve several factors which have varying weights across patients and gender. The difference between early and later onset could reflect the differential operation of two such factors. The disproportionate rate of men with early onset suggests that perhaps early-onset cases have a different or more severe disorder than later onsets, akin to the more severe early-onset form of schizophrenia often seen in men (Flor-Henry, 1985; Lorangen, 1984). Some literature suggests that early onset is a bad prognostic factor (Strauss, 1948; Ingram, 1961; Rasmussen & Tsuang, 1986) and associated with greater severity of the disorder (de Silva *et al*, 1977).

The early-onset cases might reflect the influence of brain damage or some other constitutional deficiency to which men are more vulnerable than women. There is a significant male preponderance for a number of disorders such as dyslexia, stuttering, conduct disorder, autism and Tourette syndrome, which some assume arise out of central nervous system disturbances that are genetically influenced. Various developmental differences in the brain between young men and women, especially in regard to right–left brain development, have been suggested as the cause of this differential morbidity (Taylor & Ounsted, 1972; Geschwind & Behan, 1982). Reasons put forward for the male predominance in these disorders include vulnerability of the left (dominant) hemisphere in males (Flor-Henry, 1985) and an X-linked modifying gene (Comings & Comings, 1986).

In Gilles de la Tourette syndrome the frequency of OC symptoms and the male preponderance (Nee *et al*, 1980; Pauls *et al*, 1986) may indicate an extreme form of early-onset OCD. Family studies of Tourette probands have found a high rate of Tourette cases among male relatives and of OCD among female relatives (Pauls *et al*, 1986). However, Tourette syndrome was not observed in a single family member of 70 children and adolescents with OCD (Swedo & Rapoport, 1989).

The later-onset type of OCD that is far more common in women than in men may be explained by the well known association between depression and OC phenomena (Welner *et al*, 1976; Gittleson, 1966; Videbech, 1975; Vaughan, 1976; Marks, 1987). Obsessions and compulsions emerging during the course of a depressive episode may continue long after the depression has waned.

Women in general are twice as likely as men to have depression, the peak incidence for 'neurotic' depression in women being between ages 26 and 40 (Weisman & Klerman, 1977). Neurotic depression is associated with OCD (see Rachman & Hodgson, 1980; Marks, 1987). We did not examine the timing of onset of OC symptoms relative to previous depression in our cases, but significantly more women than men had a history of depression and those with treated depression had a later age of onset. In another study (Welner *et al*, 1976), patients who developed OCD concurrent with depression or after it had a later mean age of onset (27 years and 24 years respectively) than those without past depression (age 20 years) or those who developed depression after OCD (age 17 years).

Another gender-related feature in our series concerns the excess of past anorexia in female cases of OCD. This point seems to have been ignored before our paper about it (Kasvikis *et al*, 1986), although the converse excess of symptoms in anorexics has long been known. The phenomenon has been present in other series of patients: among 150 cases (82 women) presenting with OCD, five had concurrent anorexia nervosa (Welner, 1976), and out of 14 women with OCD, three had a history of anorexia (Insel, 1984). The reason for the surfeit of past anorexia among women with OCD is unclear. It is not simply related to anxiety, since among 100 agoraphobics in our clinic, 83 of whom were women, none had a history of anorexia.

Several studies (Akhtar *et al*, 1975; Dowson, 1977; Stern & Cobb, 1978; Rachman & Hodgson, 1980) have found that obsessive–compulsive washers are more often female and those with checking rituals more often male, but this sex difference only amounted to a trend in our sample as a whole. However, in our study, type of ritual was associated with age of onset; significantly more cases with early onset exhibited checking rituals while cases of late onset showed significantly more washing rituals. Marked compulsive slowness resulting from extreme severity of disorder is much more common among men (Ratnasurya *et al*, unpublished data), but we did not examine this. In a recent study "the need for symmetry and exactness" in association with checking and counting rituals was significantly more common in men and early-onset cases (Rasmussen & Tsuang, 1986). Obsessions about numbers are more common among boys (Swedo & Rapoport, 1989) and adult men with OCD (Skoog, 1965).

The gender-related features of our series may well be found in most clinical series of OCD if they are searched for systematically. The overall demographic and clinical features of our cases with OCD were typical of those found in other series in the literature (for review see Marks, 1987). These included a slight overall preponderance of women, a low rate of marriage and of fertility, an onset mainly before age 35, a long illness duration, and frequent present and past depression.

Our cases' mean overall age of onset of 22 years and the significantly earlier mean age at onset for men accords with that in other studies (Pollit, 1957; Ingram, 1961; Lo, 1967; Welner *et al*, 1976; Coryell, 1981; Rasmussen & Tsuang, 1986). A cohort of OCD subjects identified in the ECA study (Karno *et al*, 1988), however, found a similar age of onset in men and women. These cases possibly represent a different population to the patients described in the clinical literature as only 9% had both obsessions and compulsions, 46% having only obsessions and 45% compulsions.

In conclusion, we found three gender-related features of OCD. The first was the surfeit of an anorexic history among women with OCD. The last two concern age of onset: the early-onset group had a male preponderance (perhaps reflecting organic factors), and the later-onset group a female preponderance (that may flow from depressive complications).

Whether the organic and depressive factors produce the sex difference in age of onset remains to be tested.

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The Current Literature

Can Lifestyle Changes Reverse Coronary Heart Disease? The Lifestyle Heart Trial

DAVID WHEATLEY and C. BASS

> "In a prospective, randomised, controlled trial to determine whether comprehensive lifestyle changes affect coronary atherosclerosis after 1 year, 28 patients were assigned to an experimental group (low-fat vegetarian diet, stopping smoking, stress management training, and moderate exercise) and 20 to a usual-care control group. 195 coronary artery lesions were analysed by quantitative coronary angiography. The average percentage diameter stenosis regressed from 40.0 (SD 16.9)% to 37.8 (16.5)% in the experimental group yet progressed from 42.7 (16.5)% to 46.1 (18.5)% in the control group. When only lesions greater than 50% stenosed were analysed, the average percentage diameter stenosis regressed from 61.1 (8.8)% to 55.8 (11.0)% in the experimental group and progressed from 61.7 (9.5)% to 64.4 (16.3)% in the control group. Overall, 82% of experimental-group patients had an average change towards regression. Comprehensive lifestyle changes may be able to bring about regression of even severe coronary atherosclerosis after only 1 year, without use of lipid-lowering drugs."

The summary quoted above is from an article by Ornish *et al* (1990). The present authors were invited to comment upon the study.

David Wheatley

If the mind controls the body, how can the patient with coronary heart disease (CHD) control the mind, in order to control the disease? One way suggested in this paper is to alter lifestyles to conform with the generally accepted measures designed to reduce the risk of CHD. However, these recommendations are by no means definitive and are probably not fully comprehensive, since the basic cause of the disease remains unknown. Nevertheless, any measures that can be taken to reduce risk and improve survival must be well worth considering, but in commenting on this paper, its value must be considered against the background of ignorance of the fundamental aetiology of CHD.

The authors advocate a change in lifestyle to *include*: "a low-fat vegetarian diet, moderate aerobic exercise, stress management training, stopping smoking, and group support". The authors do not say why they chose these particular interventions or, indeed, whether the lifestyle programme did in fact consist of other measures also. Diet is perhaps one of the most controversial issues in the prevention of CHD, and the diet used in this study included: "fruits, vegetables, grains, legumes and soybean products without caloric restriction". No animal products were allowed, except egg white and one cup per day of non-fat milk or yoghurt. Whether the restriction on 'animal products' included fish is not stated, although it must be presumed. This is an important omission, since Burr *et al* (1989), in an investigation of the effects of changes in fat, fish, and fibre intakes on death and myocardial reinfarction, concluded that: "a modest intake of fatty fish (2 or 3 portions per week) may reduce mortality".

Further dietary measures undertaken in the study included the elimination of caffeine and the limitation of alcohol to no more than two units per day, the latter being totally excluded for anyone with a history of alcoholism. Furthermore, "no-one was encouraged to drink". The role of alcohol in the aetiology of CHD is indeed an ambiguous one and there are a number of reports that moderate alcohol consumption does in fact exert a protective action (Klatsby *et al*, 1974; Hennekens *et al*, 1978), whereas excess is undoubtedly dangerous (Paul *et al*, 1963; Wilhelmsen *et al*, 1973). Also, qualitative differences may exist, since St Leger *et al* (1979) have shown a strong negative association between CHD deaths and alcohol, wholly attributable to wine consumption. In order to make the dietary recommendations as comprehensive as possible, it might have been better to encourage the subjects to drink moderately at the specified rate of two units daily, while making the grape their first choice of source. The elimination of caffeine from the experimental diet is to be applauded since, in a survey of 19 398 men and 19 166 women in Norway, Tverdal *et al* (1990) concluded that "coffee may affect mortality from coronary heart disease over and above its effect in raising cholesterol concentrations". The source of caffeine is not specified, but it is to be assumed that patients were warned of its presence in tea, Coca-Cola, and other drinks, as well as in coffee, since this is not always appreciated by the general public.

In their important endeavour to change lifestyle in these patients, the authors have selected a number of cardiologically relevant factors. And yet there are a number of others also that could have been considered. For example, sleep is a potent influence on CHD, as Partinen (1985) has shown. In his large cross-sectional study of 5419 Finnish men, a greater incidence of CHD was found among those who slept for more than nine hours or for less than six hours per night, as compared with those sleeping the more usual seven to eight hours. Sexual activity is not mentioned, but this may produce an adverse effect on CHD as a result of the haemodynamic changes that take place during intercourse (Hellestein & Friedman, 1970). Furthermore, sudden deaths do occur during or shortly after sexual intercourse, usually in clandestine circumstances (*British Medical Journal*, 1976). Consequently, some recommendations concerning sexual activity would have been appropriate in ensuring comparability of the experimental and control groups.

Perhaps the most surprising omission is the lack of any mention of personality factors, in view of the enormous amount of research that has been undertaken on the type A/B dichotomy in relation to CHD (Friedman & Rosenman, 1959). Although Raglan & Brand (1988) have produced evidence showing that type A individuals may actually have less CHD than type B, some classification of patients according to this factor would have been useful. There is no mention of any psychiatric assessment of these cases, and yet the CHD patient is particularly susceptible to anxiety and depression, which may exert adverse effects on the heart (Wheatley, 1981). It would have been of interest to know whether the two groups matched one another in relation to such psychiatric morbidity, which could have been assessed on standard rating scales.

And so what of the quality and reliability of the study itself? From an original sample of 193 patients, only 94 (49%) met the full entry criteria. Of these, 53 were randomly assigned to the experimental group and 43 to the control group, but of these only 28 and 20 respectively agreed to take part in the study. These numbers were further reduced to 22 and 19 respectively as a result of drop-outs during the trial. A small sample indeed, when it is stated that: "These patients represented a cross-section of age, gender, race, ethnic group, socio-economic status and educational level"!

For the experimental group, the stress-management techniques included: "stretching exercises, breathing techniques, meditation, progressive relaxation and imagery. The purpose of each technique was to increase the patient's sense of relaxation, concentration and awareness". Patients were asked to practise these stress-management techniques for at least one hour per day, and were given a one-hour audio cassette tape to assist. Twice-weekly group discussions provided social support to help patients adhere to the programme of lifestyle change.

The study lasted for one year, results being recorded on 195 coronary artery lesions by means of coronary angiography. These showed a significant regression in stenosis in the experimental group, compared with an increase in the control group. There were also significant reductions in the control group in total cholesterol, low-density lipoprotein cholesterol, and weight, but no significant differences in relation to high-density lipoprotein cholesterol, triglycerides, or blood pressure.

While there is no doubt that the authors have demonstrated improvement in some important measures of CHD in their experimental group, there are a number of missing factors. Their conclusion that: "Comprehensive life-style changes may be able to bring about regression of even severe coronary atherosclerosis after only one year, without use of lipid lowering drugs," should therefore be interpreted with caution. Nevertheless, the concept that changing lifestyles may affect prognosis beneficially in the coronary patient is of fundamental importance. Following the lead given by these authors, more comprehensive studies on larger samples should now be undertaken.

C. Bass

My first reaction on reading the report of this ambitious study was to wonder what kind of patient would undertake such a rigorous regime which, in addition to the dietary and exercise modifications, included twice-weekly group therapy? Many must have been daunted by this prospect, because, of patients who met the entry criteria for the study, only 50% agreed to take part *after* randomisation. This is a dispiritingly low figure for an intervention which was designed to improve general health and reduce morbidity from the effects of CHD. Perhaps patients were discouraged by the enormous, year-long commitment they were being asked to undertake, which began with an induction period lasting a week in a local hotel! I suspect that the eyes of south London patients with angina would glaze over when offered a treatment programme which included no caffeine, two units of alcohol per day, one hour of stress-management exercise each day, three hours each week of exercise, and twice-weekly group discussions. If only half the sample eligible accepted it in the San Francisco area, where there is greater awareness of the psychosocial contribution to morbidity from heart disease, then how applicable is this kind of intervention to other parts of the world?

There are important discrepancies between the two groups with regard to both demographic and coronary risk factors. The experimental group had received more education (15.9 v. 14.2 years), but despite this, no information about their occupational status is provided. Indeed, only one of the 28 patients in the experimental group was smoking at baseline, which confirms my suspicion that they may have been a highly selected group of non-manual workers. This is an important issue, because if there is an inverse association between social class and cardiovascular disease (Marmot *et al*, 1984), then intervention should be acceptable to and targeted at those most at risk. Furthermore, the experimental group lost over 10 kg during the course of the year, whereas the controls *gained* 0.8 kg; this weight reduction may account for improvement in anginal symptoms and possibly reduction in serum lipids.

Another important shortcoming of this study, acknowledged by the authors, is that it is not possible to state which component of the intervention led to changes in coronary atherosclerosis. The dietary intervention was clearly adhered to and led to significant reductions in cholesterol and low-density lipoprotein cholesterol. Similar findings were reported in the impressive Oslo study (Hjerman *et al*, 1981), where the incidence of fatal and non-fatal myocardial infarction and of sudden death was reduced by almost 50%. Did this important dietary change override the possible beneficial effects of exercise or stress reduction? Or could the social support of twice-weekly group therapy be the active ingredient? To answer these difficult questions would require comparison of large samples of patients, using fairly specific and 'uncontaminated' interventions. Such studies are often impractical and present enormous methodological problems. They are also very costly.

Although it is not clear which of the many components of intervention were effective, the study confirms previous findings that systematic, detailed, and personalised programmes for altering risk factors are more effective than less intensive approaches (Lovibond *et al*, 1986). The study also has none of the disadvantages associated with drug side-effects, which were considered to offset the beneficial effects of substantial reductions in smoking and serum cholesterol in the study by the Multiple Risk Factor Intervention Trial Research Group (1982).

Studies of this kind are innovative and exciting but, in my view, unlikely to make any impact on the way British cardiologists manage patients with angina. Why should this be so? In the UK, there has always been an interesting discrepancy between lay attitudes to stress and the views of cardiologists, who rarely consider it as a causal factor. Most cardiologists will tell you (and their patients) that CHD is a consequence of a sum of risk factors, but stress will rarely be cited as a contributory factor. This view has also been expressed in reports by British (but not North American) expert committees in the prevention of CHD. I suspect that one of the reasons why cardiologists give scant attention to stress is that it cannot be defined, and therefore not measured and not amenable to scientific study (Marmot, 1987).

These attitudes persist, despite the fact that impressive studies have been published which suggest that interventions designed to alter type A behaviour reduce myocardial infarction (Friedman *et al*, 1984, 1986). Despite these studies, which have received wide publicity, I am not aware of changes in the practice of cardiologists. There has been no subsequent increase in the use of group treatment following myocardial infarction, in which men would be encouraged to change those affects (anger and irritability) and types of behaviour that are thought to promote atherogenesis. Most cardiologists continue to target 'hard' risk factors such as smoking and blood lipids, and employ antihypertensive therapy – the psyche is largely ignored. I suspect that as cardiology becomes even more technologically orientated in the 1990s, behavioural approaches to treatment, like the one outlined by Ornish *et al*, will not be widely adopted. The authors acknowledge this when they remark in their discussion that "the point of our study is to determine what is true, not what is practicable." This is regrettable, because studies which consider both psychosocial and biological factors deserve more financial support.

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A Survey of Psychiatrically Ill Asian Children

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This case-note survey compares a group of Asian children referred to child psychiatry and guidance clinics in Bolton with a control group of white children referred to the same clinics. The proportion of Asian children was less than the proportion of Asian people in the general population. The proportion of Asian children coming from a broken home or a single-parent family was significantly lower than in the control group. Conduct disorders were more common in the control group and somatoform disorders were more common in the Asian group.

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The relationship of immigration to psychiatric disorders has long since been an area of interest for researchers. Among the adult population an excess of psychiatric disorders has been shown in many immigrant groups and various aetiological theories have been proposed to explain this excess, for example a misperception of cultural norms of immigrant groups, the selection hypothesis and the stress hypothesis. Similarly, an excess of psychiatric disorders has also been found in immigrant children. Many factors of aetiological significance have been identified, including a higher incidence of malnutrition in early years of life, large family size, parents working longer hours, poor and overcrowded housing, racial discrimination (Macintosh & Smith, 1974) and, in the cases of newly immigrant children, the stress of migration itself.

In Britain psychiatric morbidity in the adult immigrant population has been extensively studied (London, 1986), but surprisingly few researchers have paid attention to immigrant children. Whatever work has been done has mostly concerned West Indian children. Population-based studies indicate that West Indian children show more behavioural problems at school (Schools Council, 1970; Rutter *et al*, 1974), and that those children who do suffer from conduct disorders do not show as much emotional disturbance, and have fewer difficulties in peer relationships, when compared with native English children (Nicol, 1971). This excess of conduct disorders can at least partly be explained by the increased rate of these disorders in West Indian girls compared with the native population (Rutter *et al*, 1974).

Although Asian children constitute a major part of the immigrant child community from the New Commonwealth Countries and Pakistan, they appear to be neglected in psychiatric research. The study most quoted (Kallarackal & Herbert, 1976) suggests that behavioural problems are less common in children with Indian parents than in white English children. This lack of information about psychiatrically disturbed Asian children was the main motivation behind this retrospective survey.

Method

The study was carried out in Bolton, a town in the traditional heartland of the textile industry in the north-west of England which, in post-war years, attracted a large number of immigrant workers from the Indian subcontinent. The Asian community of Bolton can be subdivided into Gujeratis, Pakistanis, Bangladeshis and others, with the first two dominating numerically. Asian people constitute about 8% of the population served by Bolton Area Health Authority. The child psychiatric services are provided by the Child Psychiatry Department of Bolton District General Hospital and a child guidance clinic run by the same department in collaboration with the local education authority.

The study group comprised all 45 Asian children (23 boys, 12 girls) referred from 1981 to 1985. The control group comprised 72 indigenous white children (46 boys, 26 girls) referred over a randomly selected six-month period in 1981–85. Statistical analysis of the age and sex distribution of the control group showed it to be representative of all referrals during the study period (age: $\chi^2=0.22$, 2 d.f; sex: $\chi^2=0.0085$, 1 d.f.).

The following information was gathered from the case notes: sex, age, structure of the family, social class, source of referral and primary diagnosis assigned by the psychiatrist who first assessed the child at the time of referral (in five instances of neurotic disorder, enuresis had been the secondary diagnosis). All data were tabulated and tested for statistical significance using d-base 111 and SPSS computer programs.

Results

Although Asians comprise about 8% of the total population of Bolton, an estimate based on the 1981 census reveals that 14.9% of all children under five years and 9.4% of those aged 5–29 are of Asian origin (Bolton Metropolitan Borough, 1987). The number of Asian children referred during the period of study was 45, out of a total of 978 referrals.

The information about social class was lacking or unreliable in most of the cases in both study and control groups, and therefore was excluded from analysis.

In the Asian group there was an excess of boys (73% v. 64% in the control group) but this difference was not statistically significant ($P<0.39$). Similarly no significant differences were found either in the age distribution ($P<0.30$) or the source of referral ($P<0.32$).

Twenty-eight of the 72 control children were living in a broken home or with a single-parent family compared with seven out of 45 Asian children, while family composition in 16 control children was not known ($P<0.001$, $\chi^2=13.07$, 1 d.f.) (Table 1).

The proportion of Asian children suffering from conduct disorder (18%) was much less than in the control children (36%) while somatoform disorders were more common among Asian children (31% v. 11%) ($P<0.02$, $\chi^2=11.582$, 4 d.f.) (Table 2). Breaking up these data into sex categories suggested that these differences were mainly due to lower rate of conduct disorders and higher rate of somatoform disorders among the Asian boys ($P<0.01$). The diagnostic comparison of girls did not reveal any significant differences ($P<0.28$). However, even in this group there was a trend to increased somatoform disorders at the expense of neurotic disorders.

An association was apparent between conduct disorders and separated families for both study groups ($\chi^2=8.04$, 1 d.f., $P<0.01$).

Discussion

This study has revealed under-representation of Asian children in the referrals made to psychiatric clinics. This may be due to a true lower prevalence of psychiatric problems among Asian children as suggested by Kallarackal & Herbert (1976). On the other hand, it may also reflect parental attitudes and perceptions. Data on source of referral do not support the latter view, because the proportion of children referred by general practitioners (where parents have the initiative) and the school health service and hospital department (where parents do not have the initiative) were not significantly different in either group. However, the proportion of Asian children referred by the school health service was more than double that of control children. Although statistically not significant, it appears to be an important underlying trend, which corresponds with the finding of Rutter *et al* (1974) that West Indian children were more likely to present problems at school than at home. Another striking finding was the lower rate of conduct disorders among Asian children, mainly determined by lower rates in the boys. Rutter *et al* (1975) have shown a strong association between 'broken homes' and higher rates of behavioural disturbance in West Indian children. Kellarackal & Herbert (1976) found lower rates of 'broken homes' in Indian children with less behavioural disturbances. This study supports this association. The proportion of children coming from a broken home or a single-parent family in the control group was 39% (the actual proportion may even be higher because information on family structure was not available in 22% of cases) which was far higher than in the Asian group (15%). This association between broken homes and conduct disorders was further confirmed by analysis of the group as a whole. But why should the family pattern selectively affect boys, as this study shows? A closer look at the family data may provide the answer. In the control group 33% of the children were living with mothers only, as compared with 9% in the Asian group. The absence of a father is more likely to affect boys than girls because of the lack of a 'role model' (Rutter, 1975). Other reasons for a lower rate of conduct disorders in Asian children may lie in

Table 1
Comparison of family structure

Family structure	Asian children no	Asian children %	Control children no.	Control children %[1]
Mother only	4	9	24	33 (43)
Father only	2	4	3	4 (5)
Both parents	38	84	28	39 (50)
Adoptive parents	1	2	1	2 (2)
Not known	0	0	16	22

$P<0.001$.

1. Percentage distribution of family structure in the control group after excluding the 'not known' is given in parenthesis

Table 2
Comparison of diagnoses

Diagnosis	Asian children no. (%)	Asian children no. of boys	Asian children no. of girls	Control children no. (%)	Control children no. of boys	Control children no. of girls
Neurotic disorders	15 (33)	10	5	28 (39)	13	15
Conduct disorders	8 (18)	5	3	26 (36)	20	6
Hyperkinesis	3 (7)	3	0	7 (10)	7	0
Psychoses	0 (0)	0	0	0 (0)	0	0
Developmental delays	5 (11)	5	0	3 (4)	1	2
Somatoform disorders	14 (31)	10	4	8 (11)	5	3

$P<0.01$.

religious views and cultural attitudes which influence the child-rearing practices in Asian homes (Ferron, 1973), and the strong family ties and constraints on children (James, 1974).

A higher rate of somatoform disorders among the Asian children may be due to parental readiness to detect and acknowledge physical symptoms, which is a cultural characteristic and is used as an explanation for a higher rate of psychosomatic symptoms in adult Asians (Ndetei & Vadher, 1984). Alternatively, it may also mean that the psychological conflicts in the Asian children tend to manifest more in the form of somatic symptoms than neurotic or conduct disorders. However, substantiation of such a hypothesis is beyond the scope of the present retrospective case-note study.

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Human Leucocyte Antigens and Tardive Dyskinesia

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HLA status was assessed in matched groups of schizophrenics, 20 with and 20 without tardive dyskinesia. The antigen frequencies were also compared with that in a control sample. The anticipated excess of HLA-B44 was not confirmed. Pooled data displayed a shortfall of HLA-Cw7 and an excess of HLA-B44 in schizophrenics relative to population controls.

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Tardive dyskinesia (TD) is one of the most serious motor complications consequent upon antipsychotic medication, occurring in approximately 20% of subjects on these drugs (Kane & Smith, 1982). Its pathophysiology is unknown but subject to much debate.

There is no clear association of the development of TD with drug dosage or duration, nor with any particular class of antipsychotic drug (Kane & Smith, 1982). This has resulted in interest being focused on patient variables. There is evidence that increasing age, female sex, affective illness and previous brain damage potentiate the risk of developing TD (Barnes, 1988).

At present it is impossible to identify individuals susceptible to TD with any confidence. However, a report by Canoso *et al* (1986) suggests that this is possible by typing patients according to their human leucocyte antigen (HLA) type. Briefly, HLA antigens are polypeptide chains present on the surface of all nucleated cells, whose expression is controlled by the individual's HLA genotype. This is located at four loci (A, B, C and DR) on the short arm of chromosome 6. Each person has therefore a total of eight alleles at these loci, allowing considerable polymorphism. Several diseases occur preferentially in subjects with certain HLA genotypes. The best example is perhaps narcolepsy, where most subjects are HLA-DR2 positive. The situation with HLA antigens in schizophrenia is unclear (McGuffin & Sturt, 1986).

It is known that long-term treatment with chlorpromazine is associated with the development of auto-antibodies (AAB) directed against histone nuclear antigens and phospholipids. This occurs in approximately 40% of subjects receiving at least 100 mg of chlorpromazine for one year. It is more likely to occur in the presence of the HLA-B44 antigen, the relative risk being 3.55 (Canoso *et al*, 1982). As movement disorder can develop contemporaneously with the development of AAB directed against neural tissue (Sydenham's chorea being perhaps the best example), Canoso *et al* wondered whether TD was more likely to develop in HLA-B44 positive subjects. They rated orofacial dyskinesia and assessed AAB status in 66 subjects who had been previously typed for HLA-B44. They found that 16 out of their 17 subjects who were positive for both HLA-B44 and AAB had moderate to severe dyskinesia. They concluded that the presence of AAB and HLA-B44 is associated with, and can be used as a predictor of, the neurological complications of chronic chlorpromazine treatment.

There are however methodological flaws in this study. There were fewer dyskinetic subjects in the AAB-negative/HLA-B44-positive group, which Canoso *et al* ascribed to the absence of AAB. However, this group was also significantly younger and this alone could account for the shortfall in dyskinesia. Also, the AAB-positive HLA-B44-positive group contained an excess of subjects with diagnoses of affective illness and organic states, and these are risk factors for the development of TD. Therefore it would appear that there are potentially powerful confounding variables which cast doubt on the conclusions of the study.

We have sought to replicate and extend this study but with better-matched groups. To that end we planned to assess the HLA type in matched groups of schizophrenic subjects differing significantly only for the presence or absence of dyskinesia. We have not assessed AAB status.

Method

Twenty subjects and 20 controls were recruited from the long-term wards at Leverndale Hospital, Glasgow. All satisfied DSM-III criteria for schizophrenia (American Psychiatric Association, 1980), were of similar ethnic origin and consented to assessment. Dyskinetic movements were rated using the Abnormal Involuntary Movement Scale (AIMS; National Institute of Mental Health, 1976) and Rockland scale (Simpson *et al*, 1979). Since dyskinetic movements can be voluntarily inhibited and exacerbated by anxiety, part of the assessment was undertaken when the subjects were unaware that their movements were being rated. During this the subjects were distracted by attempting to memorise a picture for two minutes. All subjects were seen twice, one week apart, by both authors, and an average score was taken. For inclusion, the dyskinetic subjects had to satisfy the criteria of Schooler & Kane (1982). The controls were required to have current, and at least five years' cumulative exposure to antipsychotic medication, and to have no dyskinetic movements at both evaluations. A change in medication in the preceding months and known neurological disease led to exclusion. All patients had current or previous exposure to chlorpromazine. The groups were matched for age, sex, length of exposure to antipsychotic medication, length of stay in hospital, and receipt of anticholinergic drugs. Current medication, in chlorpromazine equivalents (Davis, 1976), was noted.

The HLA typing was undertaken using the NIH method (Teresaki, 1970). In addition to the comparison with non-dyskinetic subjects, antigen frequencies in the dyskinetic group were compared with those in an unrelated British control panel of 500 subjects. The age structure of the latter control panel is unknown.

Antigen frequencies were compared between the two schizophrenic groups using Fisher's exact test, and between the dyskinetic group and the control panel using the χ^2 test with Yates' correction where indicated. Significance levels were corrected using the method of Edwards (1974). The relative risk (RR) was calculated by the method of Woolf as modified by Haldane (1955).

Results

Table 1 shows that the groups were well matched. No significant differences were found in the antigen frequencies between our dyskinetic and non-dyskinetic groups. In particular HLA-B44 was found in six of 20 dyskinetic subjects compared with 10 of 20 controls. A significant shortfall of HLA-Cw7 was found between our dyskinetic subjects and population controls (0% v. 42.7%, $P=0.0005$, P corrected = 0.0139, RR = 0.0328, 95% confidence intervals = 0.0095–0.532).

Table 1
The matching of the study and control schizophrenics

	Subjects	Controls	Student's *t*
Age. years			
mean (s.d.)	59.4 (6.3)	60.9 (6.8)	0.649
range	45–70	44–72	
No. of men/women	12/8	12/8	
Mean (s.d.) length of illness: years	31.2 (7.8)	30.8 (7.4)	0.226
Mean (s.d.) stay in hospital: years	21.8 (15.9)	23.8 (15.6)	0.286
Mean (s.d.) daily dosage of medication: mg chlorpromazine equivalents	358 (394)	533 (174)	1.329
No. taking anticholinergic drugs	9	8	

No significant differences.

We also found an excess of HLA-B44 in the pooled data of schizophrenics compared with population controls ($P = 0.005$, P corrected = 0.225, RR = 3.02, 95% confidence intervals = 0.367–24.77). (Full data are available on application to the authors.)

Discussion

We have been unable to replicate the findings of Canoso *et al* (1986). However, our sample size was not large enough to exclude a relationship between HLA-B44 and TD: we have calculated that, if TD is present in 20% of the HLA-B44-negative subjects, and if the HLA-B44 antigen doubles the risk of developing TD (roughly in line with Canoso's findings), the sample size required to detect an association at the 5% significance level would be 115. However, the trend was not in that direction, the HLA-B44 antigen being almost twice as common in our non-dyskinetic controls than in our subjects. An association between dyskinesia and HLA-D/DR antigens remains a possibility.

There was a shortfall of HLA-Cw7 in our dyskinetic subjects compared with our population controls. However, these two groups also differed with regard to the presence or absence of schizophrenia. Furthermore the shortfall persisted in the pooled groups, suggesting that the relationship may be with schizophrenia rather than with tardive dyskinesia. We are unaware of any other reports suggesting an association between HLA-B44 and schizophrenia and therefore this result requires confirmation. Our finding of an excess of HLA-B44 in schizophrenia may seem to bridge our study with that of Canoso *et al*, but scrutiny of their data with regard to diagnosis did not confirm this.

A methodological problem in the present study would appear to be the possible discrepancy in age structure between the subjects and the population control panel. However, no difference in HLA profile has been noted with ageing (Blackwelder *et al*, 1982).

A major difficulty in recruiting controls in studies of TD is that an unknown proportion of the controls may later develop TD. As most subjects developed TD within five years of commencing antipsychotic drugs, one solution is to recruit controls who have survived this period without developing TD. Our controls had been on medication for a mean of 30.8 years, making it unlikely that any would later become dyskinetic.

TD may not be a unitary condition. There is some evidence both for topographical (orofacial v. limb–truncal) and temporal (early v. late-onset) subtypes (Barnes, 1988). Their aetiology and pathophysiology may differ. Unfortunately Canoso *et al* assessed only orofacial dyskinesia. We found no differences in HLA-type between orofacial and limb–truncal TD, but the numbers in each group were small, precluding any firm conclusion.

In conclusion, we have found that HLA-B44 has neither the required specificity nor sensitivity to be a useful predictor of the neurological complications of antipsychotic medication.

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The Neuropsychiatric Evolution of a Case of Progressive Supranuclear Palsy

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The neuropsychiatric changes in a Chinese patient suffering from progressive supranuclear palsy led to a difficult diagnosis. Polysomnography revealed sleep fragmentation and central apnoea.

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Progressive supranuclear palsy (PSP), or Steele-Richardson–Olszewski syndrome, is a rare multi-systemic neurodegenerative disorder, of unknown aetiology and of the presenium, which consists of the clinical triad of gait disturbance, mental change and the almost pathognomonic sign of nuclear/supranuclear ophthalmoplegia (particularly involving vertical gaze). Associated features include axial dystonia in extension, rigidity, pseudobulbar palsy and less constant pyramidal and cerebellar signs. The main neuropathic finding is histologically distinct neurofibrillary tangles and neuronal loss with gliosis in the diencephalic, brain-stem and cerebellar nuclei (Steele *et al*, 1964; Brusa *et al*, 1980; Kristensen, 1985). Although it is a distinct clinicopathological entity, misdiagnosis is common (Perkin *et al*, 1978).

The literature on PSP has been neurologically orientated (Kristensen, 1985). Mental changes have been ignored (Perkin *et al*, 1978), or only cursorily mentioned, such as "irritability, suspiciousness, a peculiar jocularity, mild personality change, confusion at times" (Steele *et al*, 1964).

Case report

Mr A, a 67-year-old retired Chinese mechanic, was referred with a five-year history of progressive personality change, deafness and later a slight limp. He was previously a hard-working and even-tempered person, but had become suspicious and irritable since his retirement seven years ago. A general practitioner diagnosed mild hypertension and gave simple reassurance only. There were frequent conflicts with family members, who believed he was malingering. At a psychiatric clinic, a diagnosis of adjustment disorder was made.

Two years later, he had the delusion that his wife would steal his money, and he threw objects at her. After admission, he was fully conscious, partially deaf, relevant in speech, with no psychotic features or cognitive deficits. Apart from a slight limp on the right, physical examination and laboratory tests, including blood copper and ceruloplasmin, and skull X-ray were normal. Because of his history of hypertension and minor stroke and an electroencephalogram (EEG) showing mild slow-wave changes, he was thought to have vascular dementia and a personality problem, and discharged.

In the next two years, his outbursts became more serious. He attempted to pour boiling water over his wife and ordered her to jump from the window. Examination showed normal cognitive function, the delusion towards his wife and a mild limp. A diagnosis of paranoid psychosis and possible deafness was made. He was given chlorpromazine (100 mg/day) with little effect. An EEG showed brief bitemporal slow (theta) waves, while a brain computerised tomography scan demonstrated moderate ventricular dilatation suggestive of cerebral atrophy, mild basal ganglia calcification and a normal cerebellum. As behavioural advice on handling his temper outbursts was ineffective, the clinical psychologist felt that "his hearing was selective, and his paranoid ideas over finance were not totally unfounded". An audiogram, however, confirmed severe neurosensory deafness.

In the following one year, the patient became more clumsy, with frequent falls and bruising. Physical examination showed a worsening limp and rigidity which were attributed to neuroleptic treatment. Six months later, however, an independent neurological reassessment revealed a stiff and deeply furrowed face, dysarthria, bradykinesia, axial and left-sided cogwheel rigidity, paralysis of volitional vertical gaze, generalised hyper-reflexia, but no Babinski or cerebellar signs, tremor or sensory deficits. His signs persisted after the neuroleptic was withdrawn, and a diagnosis of PSP was made. The rigidity improved only slightly with carbidopa (275 mg/day) and benzhexol (6 mg/day), while respiratory arhythmia and mild cognitive deficits slowly emerged. His paranoia changed into dependence on his wife.

A nocturnal polysomnogram showed an actual sleep time of six hours and a fragmented sleep architecture, consisting of very little slow-wave sleep (SWS) and little rapid eye movement (REM) sleep (stage 1: 1 h, 34 min (25.5% of actual sleep time); stage 2: 3 h 48 min (61.7%); stage 3: 4 min (1.3%); stage 4: nil; total REM: 42 min (11.5%)). A total of 148 apnoeic and 16 hypopnoeic episodes, predominantly of central origin, were detected. These were associated with oxygen desaturation of 86% and a longest duration of 84 seconds. The apnoea index (this is the number of apnoeic episodes per hour of sleep, and is not more than five in normal adults) was 26.6. There was no obstructive element from nasopharynx to larynx. The addition of amitriptyline (50 mg *nocte*) led to modest improvement in his clumsiness. With referral to a nearby geriatric day centre, his relatives were able to take care of him at home.

Discussion

Early diagnosis of PSP is difficult because its symptoms are easily dismissed as features common to the elderly. The initial label of an adjustment disorder led to inappropriate treatment and reinforced family conflicts. Paranoid symptoms and irascibility were not uncommonly mentioned in previous reports which ignored otological assessment (e.g. Steele *et al*, 1964; Perkin *et al*, 1978; Brusa *et al*, 1980; D'Antona *et al*, 1985). However, one study revealed varying degrees of perceptive deafness in all eight audiometrically tested patients (Dix *et al*, 1971). In two out of three patients, Perkin *et al* (1978) detected bilateral high-tone loss and perceptive deafness – the latter patient presented with paranoid accusations of his wife trying to poison him. The association of deafness with paranoid symptoms therefore deserves closer examination in PSP. Unlike patients with untreated functional delusional disorders, paranoid symptoms are less organised and may spontaneously lessen in PSP patients, as with our patient. This difference in prognosis should be clearly explained to distressed relatives.

Personality changes were cursorily noted to be "a confirmed hallmark of PSP from onset" (Brusa *et al*, 1980), but were usually not specifically described. The author suggests the DSM–III–R diagnosis of organic personality syndrome, which consists of "a change in personality style and behaviour manifested primarily by the impaired control of the expression of emotions and impulses" (American Psychiatric Association, 1987). This may indicate frontal lobe dysfunction, which has been demonstrated both neuropsychologically (Maher *et al*, 1985) and by concurrent studies with positron-emission tomography (D'Antona *et al*, 1985). When the patient was deluded, organic delusional syndrome should be diagnosed (American Psychiatric Association, 1987). With the manifestation of cognitive and functional deterioration, a diagnosis of dementia should then be made. This is typically variable and may be characterised by personality, emotional or perceptual changes instead of cognitive decline (Kristensen, 1985). Indeed, Steele *et al* (1964) wrote that "dementia with changes in personality and behaviour was often early . . . but tended to remain mild," and that "a lengthy holiday failed to improve the situation" of their patient 1 who became "very discouraged, impatient and frustrated". This paralleled ineffectve psychological treatment for our patient.

Given the widespread neuropathological changes in PSP (Steele *et al*, 1964; Brusa *et al*, 1980), clinical diversity is expected (Kristensen, 1985). Although Steele *et al* (1964) described ophthalmoplegia to be "constant, striking and usually early," lack of awareness and late appearance contributed to diagnostic delay in our patient. The paralysis of downward gaze is due to lesions involving the lateral portion of the rostral interstitial nucleus of the medial longitudinal fasciculus (Daroff, 1987). Even in the absence of clinically overt oculomotor dysfuncion, detailed electronystagmography (ENG) and oculography (EOG) performed at an earlier stage may be diagnostic (Kristensen, 1985). Unlike speech and gait problems, which may improve with benztropine, ophthalmoplegia retains its diagnostic value even in drug-treated patients.

The patient's increased rigidity was assumed to be induced by neuroleptics. Mild relief with anticholinergic drugs served to reinforce this self-fulfilling hypothesis. Thus, caution should be exercised in using neuroleptic and anticholinergic drugs in suspected PSP patients, as they may mask or worsen extrapyramidal symptoms, to which they may be more prone. Besides, if PSP, like Parkinsonism, is due to dopaminergic deficiency, neuroleptics may contribute to neurological deterioration or unmask an otherwise subclinical PSP because of their antidopaminergic activity. However, the poor response of most PSP patients to carbidopa or benzhexol argues against such a hypothesis, and the neurochemistry of PSP remains a puzzle (Kristensen, 1985).

The polysomnograph indicates disorganisation of sleep-regulating centres. The raphe nuclei (containing serotonergic neurons and believed to regulate non-REM sleep) and the locus coeruleus/subcoeruleus (containing noradrenergic neurons and believed to govern REM sleep) are both areas of potential neuropathic change in PSP (Kristensen, 1985), so that disorders of arousal and sleep may be expected. In PSP patients, EEG patterns characteristic of wakefulness have been found during behavioural sleep, suggesting that mechanisms accounting for EEG patterns and the behavioural manifestations of sleep may become uncoupled. The patient suffered from sleep deprivation and a moderate degree of central sleep apnoea with oxygen desaturation, both of which can cause neuropsychological dysfunction and probably psychosis. Instead of loud snoring and excessive daytime sleepiness, central sleep apnoea may present with mild tiredness, insomnia and frequent wakings, which have been reported in PSP patients (Sadler *et al*, 1984). It is worth investigating whether improvement in PSP patients put on low-dose amitriptyline (Yamashi *et al*, 1986) is related to the latter's known effect on reducing sleep apnoea (Lee *et al*, 1989).

Thus, PSP should be considered in the differential diagnosis of late-onset personality change, and continued neurological assessment for ophthalmoplegia and movement disorder are important for accurate diagnosis and treatment.

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Trazodone-Induced Transient Hypomanic Symptoms and their Management

S. JABEEN and C. J. FISHER

Transient trazodone-induced hypomanic symptoms occurred in three depressed patients. Trazodone therapy was continued or reinstated shortly after the hypomanic episode without further evidence of hypomania.

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Trazodone is a triazolopyridine derivative with established antidepressant activity (Feighner & Boyer, 1988). It is chemically unrelated to known psychotropic drugs and has a pharmacological profile unlike that of conventional antidepressants. It is more selective than tricyclic antidepressants in its ability to inhibit synaptosomal uptake of serotonin, and is unusual in that it is a serotonin antagonist at low doses and an agonist at high doses (Maj *et al*, 1979). It also blocks pre-synaptic α_2-receptors, thereby increasing noradrenaline release. Trazodone reduces post-synaptic β-adrenergic receptor sensitivity after chronic but not acute administration (Clement-Jewery, 1981), an effect similar to that produced by tricyclic antidepressants. It has minimal anticholinergic side-effects. On the other hand, sedation and postural hypotension seem to be frequent.

In the USA, seven cases have been reported to develop a clinical picture consistent with a DSM-III diagnosis of manic or hypomanic episode when treated with trazodone.

We report three patients who developed transient hypomanic features and discuss the management of these patients.

Case reports

Case 1

A 61-year-old man presented with a six-month history of panic attacks in the context of long-standing anxiety symptoms. He had been taking chlordiazepoxide (7.5 mg daily) and temazepam (20 mg daily) for many years.

There was no past or family history of affective disorder. He was admitted for benzodiazepine withdrawal, during the first week of which he developed severe depression, with biological symptoms, and became suicidal. He was prescribed trazodone (100 mg *nocte*). On the fourth day of treatment he became cheerful, garrulous, overactive and exhibited pressure of speech. Trazodone was stopped and within two days he became depressed and suicidal again. Trazodone was restarted and dosage later increased to 200 mg. Lithium was added and the dose was increased to obtain therapeutic serum levels. His mood started improving after one week. Benzodiazepines were gradually withdrawn and he was discharged three weeks later and has remained well.

Case 2

A 24-year-old woman was admitted to hospital with post-natal depression with biological features and suicidal ideation. She had had two previous episodes of depression after her father's death and after the birth of her first child, but had not received psychiatric treatment. Two of her siblings had suffered from unspecified psychiatric illnesses which had required in-patient care.

She was started on trazodone (100 mg per day, increased to 150 mg after three days) and on the fourth day her mood became elated – she was unable to stop laughing and became overactive. Trazodone was decreased to 100 mg per day and she became depressed the following week. Trazodone was increased to 150 mg without any hypomanic swing and she was discharged from hospital and maintained her improvement at out-patient follow-up.

Case 3

A 70-year-old man presented to the out-patient department with depression and an inability to go out. Previous episodes over 17 years had responded to standard antidepressants. There was no history of manic episodes or any family history of mental illness. He was prescribed trazodone (50 mg twice a day) and, on review after four weeks of treatment, reported dramatic improvement: he felt "too well", became overactive, dismissed his homehelp and began to go out more. In the light of our experience with the two cases above, we increased the dose to 50 mg three times a day to reach the lower end of the recommended therapeutic dose range, and he was carefully monitored. During the following six weeks his hypomanic symptoms resolved and he became mildly depressed; trazodone was increased to 200 mg per day and his mood improved without any hypomanic features.

Discussion

A recognised side-effect of tricyclic antidepressants and monoamine oxidase inhibitors is hypomania or mania in bipolar or unipolar depressed patients. A 'switch' rate of 8.5% with the use of tricyclic antidepressants and 11.1% with the use of mono-amino oxidase inhibitors has been suggested (Bunney, 1978). The switch to mania seen with trazodone suggests that trazodone may have an effect on those putative aminergic systems involved in the switch process. All seven cases reported previously are described briefly in Table 1 for comparison. Time of onset after initiation of trazodone treatment, severity of symptoms and management varied from case to case and only five of the cases progressed to full-blown mania. In five out of the ten total cases, hypomanic symptoms developed very early in the course of treatment, well before normal antidepressant effects are usually seen. Emergence of hypomanic symptoms at an early stage of treatment could be due to a sudden rise in synaptic serotonin and noradrenaline levels.

Our patients differ from previously reported cases in that they did not develop full-blown hypomania. In two cases the elevated mood responded to stopping or reducing the dose of trazodone, but only to be followed by recurrence of depression which later responded to trazodone at the same or increased dose without further evidence of hypomania. In the third case trazodone was continued in an increased dose and the hypomanic symptoms settled. It is apparent that the hypomanic effect of trazodone can occur in the absence of any past or family history of hypomania. (The UK manufacturers, Roussel, and the Committee on the Safety of Medicines have been informed of these three cases, but neither is aware of any other cases.)

Our experience suggests that trazodone-induced hypomanic symptoms may be transient and that immediate withdrawal of trazodone may not always be necessary. Hypomanic symptoms may respond to dose reduction and, if the depression recurs, this may respond to trazodone without further hypomanic swings. Lithium may not be required in these patients.

We suggest that this complication of trazodone therapy is variable and these patients do not represent a homogeneous group. Further experience is needed before firm conclusions about management can be drawn.

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Table 1
Summary of clinical features and management of previously reported cases

References	Age and sex	Initial diagnosis (DSM-III)	Trazodone dose per day	Duration of treatment at onset: days	Clinical features	Diagnosed as (DSM-III)	Management and results
Escobar (1980)	Middle-aged man	Depressed	400 mg	14		Mania	
Warren & Bick (1984)	57-year-old woman	Major depression	150 mg	7	Agitated, losing contact with reality, pressure of speech, hyperactivity, insomnia, grandiose ideas and delusions	Mania	(a) Therapeutic dose of lithium – no improvement (b) Thiothixene 40 mg daily – slight improvement after one week (c) Trazodone discontinued at this stage – return of pre-morbid state within 2 days
	25-year-old woman	Major depression	400 mg	30	Acute paranoia, hyper-irritability, inability to sleep, impulsivity, inflated self-worth	Mania	(a) Trazodone reduced to 300 mg and then 200 mg/day with no change in symptoms. Chlorpramazine 200 mg/day was added, to no effect (b) Trazodone discontinued, patient started to settle down over next 10 days, no depression on withdrawal
Arana & Kaplan (1985)	47-year-old woman	Major depression	400 mg	17	Paranoid delusions, auditory hallucinations, loosening of association, dysphoric mood and irritability	Mania	Trazodone discontinued, within two weeks recurrence of depression was noted
	66-year-old man	Major depression	100 mg	4	Agitated, hyperactive, pressure of speech, labile affect, racing thoughts, marked insomnia and irritability	Mania	Trazodone discontinued and 2 days later patient was depressed
Mann (1981)		Major depression				Hypomania	Trazodone discontinued, hypomania subsided spontaneously
Lennhof (1987)	53-year-old man	Major depression	150 mg	4	Insomnia, restlessness, hyperactivity, pressured speech, irritability, racing thoughts, elation and grandiosity	Hypomania	Trazodone decreased to 500 mg/day and lithium carbonate (300 mg) was added. Within one day, hypomanic state remitted

Not all details given in reports

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Encephalopathy Resembling Creutzfeldt–Jakob Disease Following Oral, Prescribed Doses of Bismuth Nitrate

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A patient given bismuth nitrate in the recommended dose range over 16 months developed panic attacks and a major depressive syndrome and, after 15 months, a myoclonic encephalopathy resembling Creutzfeldt–Jakob disease. He recovered four months after bismuth had been discontinued. Bismuth preparations are becoming popular in the treatment of gastritis; close attention to neurological and psychiatric symptoms seems mandatory.

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Bismuth salts and preparations have been traditionally used in the treatment of haemorrhoids, functional dyspepsia and other non-specific gastrointestinal disorders. In Germany and the US commercially available products include bismuth oxyiodite, bismuth carbonate and bismuth gallate (haemorrhoidal suppositories and creams) as well as bismuth salicylate and nitrate (oral preparations). Despite numerous reports of acute myoclonic encephalopathy after long-term use of insoluble bismuth salts, this type of therapy has recently become popular in the treatment of peptic ulcers and chronic gastritis, because of its postulated bactericidal effect on campylobacter (Boersch, 1988). The concept of parenteral non-absorption of bismuth salts can hardly be maintained: bismuth-induced encephalopathy has been described after 10 days of treatment (Bouche & Derouesne, 1977) and with blood levels well below therapeutic recommendations (Monstrey *et al*, 1978).

We report one case of acute myoclonic encephalopathy with prodromal symptoms starting two months after therapy with bismuth nitrate had been initiated and first diagnosed as panic attacks and major depressive syndrome.

Case report

A 69-year-old retired engineer was prescribed a commercial preparation of granulated bismuth nitrate (8 g/day p.o.) (the recommended dose in Germany) over three weeks, then 4 g/day p.o. over 16 months, for non-specific gastrointestinal complaints. His first symptoms developed after two months: malaise, nausea, dizziness, night-sweats and dry mouth. Over the following six months he experienced depressed mood, marked insomnia, lack of interest and loss of pleasure in most activities, fatigue, loss of energy, diminished ability to concentrate and indecisiveness. In addition, he developed typical panic attacks predominantly at night, with dizziness, trembling, sweating and fear of dying. Between attacks there was increasing nervousness and apprehension.

Twelve months after initiation of bismuth therapy, he was diagnosed as suffering from a major depressive syndrome and was treated with tricyclic antidepressants and benzodiazepines. Both depressive symptoms and panic attacks improved. Shortly thereafter, he began to complain of cognitive and neurological deficits: problems with impaired digit span and short- and long-term memory, difficulty reading and writing, tremor and vertigo, dysarthria and reduced arithmetic skills. After 15 months the full clinical syndrome of a myoclonic encephalopathy became evident:

(a) progressive dementia with impairment in short- and long-term memory, abstract thinking and judgement, which significantly interfered with his usual social activities
(b) intermittent delirious states with reduced ability to maintain attention, disorganised thinking, perceptual disturbances and insomnia
(c) dysarthria
(d) progressive ataxia
(e) tremor and myoclonus of the upper extremities.

The electroencephalography (EEG) findings were not specific (increased beta activity, without periodic spike and wave patterns). Studies of sensory-evoked potentials showed giant potentials after cortical stimulation. Cerebrospinal fluid (CSF) and routine laboratory studies, electromyelography, Doppler sonography and toxicology screens for heavy metals were all normal. The clinical syndrome of a rapidly progressive cognitive impairment, combined with neurological symptoms like ataxia, myoclonus and giant sensory-evoked potentials and a rapid clinical deterioration led to a preliminary diagnosis of Creutzfeldt-Jakob disease, although typical triphasic EEG potentials were never found.

At this point it became known that the patient had been taking bismuth preparations during the past 15 months. However, a urine toxicology screen for bismuth taken five days after bismuth was discontinued had been negative. The patient was treated with steroids by parenteral infusion, improved rapidly and was well four months later.

Discussion

The clinical syndrome of a subacute, progressive dementia with intermittent delirious states and myoclonus, disturbances of gait and co-ordination, and all manner of mental aberration in a middle-aged man, may be a manifestation of Creutzfeldt-Jakob or Heidenhain's disease (Adams & Victor, 1981), but has also been described in relation to bronchogenic carcinoma, benign post-infectious (possibly viral) illness (Baringer *et al*, 1968), viral encephalitis and intoxication with methyl bromide or bismuth (Adams & Victor, 1981). An inflammatory or infectious process could initially be excluded since all blood and CSF studies remained within normal limits. In addition to the symptoms described above, this absence of pathological laboratory findings and the appearance of periodic giant potentials in the EEG evoked by sensory stimulation led to a preliminary diagnosis of Creutzfeldt-Jakob disease. Clearly, this disorder can now be excluded on grounds of the benign course, which holds true for a neoplastic process and Heidenhain's disease as well. Tricyclic antidepressants may at times in elderly people induce insomnia, agitation or delirious syndromes; however, they do not lead to a progressive dementia.

The ingestion of bismuth nitrate over 15 months, the full recovery after bismuth had been discontinued, and the clinical picture favour the diagnosis of a bismuth encephalopathy. However, according to the manufacturer's statements, resorption of bismuth nitrate from the gastrointestinal tract should occur only where there is extensive inflammatory bowel disease, which had been ruled out by previous gastroscopy and colonoscopy. Urine bismuth levels proved normal.

Our diagnoses were then: (a) bismuth mood disorder, depressed (293.83, DSM-III-R; American Psychiatric Association, 1987), (b) bismuth anxiety disorder (294.80, DSM-III-R), (c) bismuth dementia (294.10, DSM-III-R), and (d) bismuth delirium (293.00, DSM-III-R).

According to Krüger (1984) and Buge *et al* (1981), this well recognised disorder runs a fairly typical course:

(a) a prodromal phase, lasting weeks or months, with prominent mood syndromes and cognitive deficits (typically, our patient was initially diagnosed with a major depressive syndrome)
(b) relatively sudden appearance of delirious states, progressive dementia, dysarthria, aphasia/ataxia, tremor and myoclonus
(c) common EEG findings of monomorphic, stable 4-6 c/s activity bilaterally in the temporo-fronto-rolandic regions, unaffected by eye opening and by photic stimulation (Viterbo *et al*, 1977). Buge *et al* (1981) suggest that bismuth blood levels above 1.5 μg/ml may, through "cortical inhibition", suppress paroxysmal features during myoclonus (Supino-Viterbo *et al*, 1977)

The unusual features of this case are the onset of symptoms following oral, prescribed doses of bismuth nitrate, in the absence of extensive inflammatory bowel disease, and with a negative urine screen for bismuth. This seems particularly relevant to clinical practice because of the recent increase in use of bismuth against campylobacter. Attention should be directed towards psychiatric or neurological symptoms even during therapy within the recommended dose range.

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Hypersexuality, Fetishism and Multiple Sclerosis

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Hypersexuality and fetishism appeared in a patient with multiple sclerosis whose MRI scan showed frontal and temporal lesions. The hypersexuality may have been the presenting symptom of the multiple sclerosis.

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Sexual activity varies according to life circumstances, but certain conditions can cause abnormal hypersexuality. Psychiatric disorders, drugs and neurological abnormalities may be implicated, and it has been reported post-ictally in temporal lobe epilepsy (Blumer, 1970) and following basal frontal lesions (Miller *et al*, 1986).

Fetishism involves the need to use inanimate objects or part objects for sexual arousal. It is likely that it has a multifactorial cause with psychodynamic, behavioural, cultural and biological components. Biological abnormalities are rare, but fetishism has been reported in association with damage to the temporal lobe and temporal lobe epilepsy (Epstein, 1961).

Hyposexuality is common in multiple sclerosis. Cases of hypersexuality have been reported (Langworthy *et al*, 1941), but without reference to any lesion. An association between multiple sclerosis and fetishism has not been previously reported.

We report a patient with multiple sclerosis who developed hypersexuality, disinhibition and a fetishism. Magnetic resonance imaging (MRI) showed lesions in the frontal and temporal lobes.

Case report

Mr A had a healthy childhood and married in his late teens. Marital breakdown followed the sudden onset of persistent demanding and promiscuous behaviour. This preceded by two years the onset of complete but temporary quadraplegia and may have been the presenting symptoms of his multiple sclerosis. He maintained an excellent work record until his paralysis forced his retirement at 25. At 26 his hypersexuality increased and he was disinhibited and assaultative. At the time his erection was good but he was unable to ejaculate. At 28 he developed a foot fetish and his hypersexuality led to offending, sometimes within sight of a policeman. Treatment in a neurology unit, the community and an ordinary psychiatric unit failed. He was imprisoned, but later admitted to a medium-secure unit. Treatments for his hypersexuality and increasingly associated aggressive behaviour included counselling, behavioural therapy, antilibidinal drugs (cyproterone acetate), neuroleptics and carbamazepine. Slight improvement often followed the introduction of a new treatment but was never sustained.

When he left the secure unit unescorted he would be found on another ward with a female patient. Having persuaded her to remove her shoes and stockings he would fondle and kiss her feet. Female visitors to the ward sometimes found themselves in similar situations. Finally a more serious assault on a female patient in the secure unit led to his transfer to a maximum-security hospital.

The illness has been progressive and at the time of reporting he had bilateral optic atrophy, bilateral extensor plantars and a broad-based ataxic gait. He is often incontinent at night and occasionally by day. Over the last year he has become impotent. He now presents as a likeable, co-operative and slightly euphoric man with an unrealistic view of his future. He is manipulative and has occasional outbursts of temper. Psychological assessments showed a decrease in his IQ over the years and suggested deficits of the left temporal lobe, right hemisphere and frontal lobe. An MRI scan shows periventricular and frontal damage (Fig. 1).

Discussion

The patient has diffuse brain damage but the main features are those of frontal lobe syndrome with damage to the temporal regions. Disinhibition occurs in the frontal lobe syndrome. While hypersexuality can occur, it is not characteristic. It has been associated with the cessation of temporal lobe seizures, either post-ictally or following a temporal

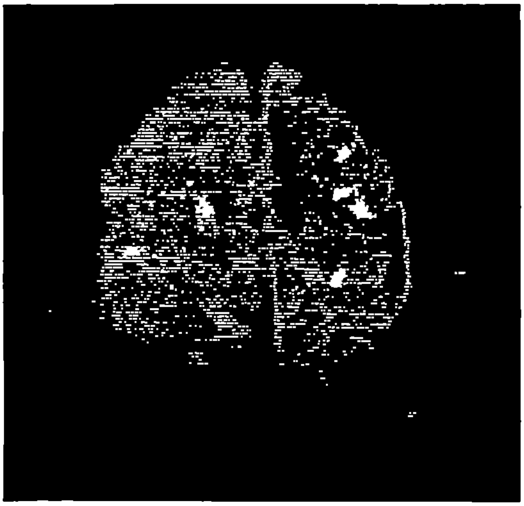

Fig. 1 The MRI scan, showing periventricular and frontal lesions.

lobectomy (Blumer, 1970). Late-onset fetishism, however, has been associated specifically with temporal lobe epilepsy and this has been shown to improve with surgical removal of the focus (Mitchel *et al*, 1954). The coexistence of disinhibition, hypersexuality and fetishism in this patient leads to the conclusion that both frontal and possibly two distinct temporal lobes pathways are involved. This would partly explain the difficulties in treating him.

The alternative to hospital care would have been imprisonment. It is perhaps a sad indictment of other health service provisions that he remains under maximum security, with possibly his greatest hope of release being a further physical deterioration.

Acknowledgement

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Editor: Ian Pullen

Contents: Prescribing in mental handicap hospitals/Buspirone in benzodiazepine withdrawal/Anorexia nervosa across cultures/Anorexia nervosa in the elderly/Psychological sequelae of torture/British and Australian depression revisited/Urinary chromatographic profiles in psychiatric diseases/Diazepam abreaction/Racial stereotypes/Stress and puerperal psychosis/Erotomania in relation to childbirth/Effects of beliefs on grief/SLE and multi-infarct dementia

Prescribing in mental handicap hospitals

SIR: Wressell *et al*'s study (*Journal*, July 1990, **157**, 101–106) has highlighted the common problems of drug prescription in mental handicap institutions, namely overprescription, polypharmacy, irrational prescription practice (e.g. antipsychotics prescribed to those with a diagnosis of neurosis) and inadequate review leading to unnecessarily prolonged drug treatment. However, I wondered why they restricted their survey to the antipsychotic drugs, ignoring other psychoactive drugs such as sedatives, anticonvulsants, antiparkinsonian drugs and antidepressants, etc. All these drugs are frequently prescribed for the mentally handicapped and they share many common side-effects. Furthermore, improper drug use like prolonged use of anticholinergic drugs, which is due to inadequate review, is known to expose the mentally handicapped population to higher risk of tardive dyskinesia.

Also, why did they not study all patients on antipsychotic drugs in 1982? Although they have shown that there was significant reduction in the dosage and polypharmacy of those patients who were given drugs in both 1982 and 1986, one important indicator of the overall improvement in drug-prescription practices would be the number of patients who could be successfully weaned off their drug treatment. Hence the authors have not provided us with adequate proof of the improvement in overall drug prescription.

A similar study (Fan, 1988) in Hong Kong yielded very similar results. A survey of all drug prescriptions for the 200 inmates of the Siu Lam Hospital who were moderately or severely mentally handicapped revealed that 69 (34.5%) were on antipsychotic drugs, 73 (36.5%) on anticonvulsants and 20 (10%) on both; 34 (17%) were on antiparkinsonian drugs, 14 (7%) on depot preparation and one on lithium. Polypharmacy was noted in 14% of inmates on antipsychotics and in 15% of inmates on anticonvulsants. In all except one case, antipsychotic drugs were prescribed for behaviour problems without any formal psychiatric diagnosis. Too frequent drug administration and inadequate review were also noted.

Comparing the findings of these two studies reveals a few points of interest. Fan found that significantly more male inmates (43%) received antipsychotic drug prescriptions than female inmates (26%) ($P<0.01$). This is compatible with the finding of Dr Wressell *et al*, and is probably related to the significantly higher prevalence of behaviour problems in the male sex ($P<0.0005$).

Secondly, although the mean daily dosage of antipsychotic drugs for the female patients (568 mg, s.d. = 777) is also higher than that of the male patients (303 mg, s.d. = 374), again compatible with Dr Wressell *et al*'s finding, it is not statistically significant (analysis of variance, $F=3.512$). When Dr Wressell *et al*'s claim of significant difference between the mean daily antipsychotic dosages of the male and female patients was subject to more critical examination, it turned out that they had miscalculated the result of the analysis of variance and hence wrongly stated the result. Recalculation of the analysis of variance with the provided data shows that the variance ratio (F) was 1.562 and hence is not statistically significant. This is not unexpected, as the standard deviations of the daily dosages are very great in both studies, especially in the case of the female patients. Therefore the apparent difference in the mean daily dosages between the male and female patients is not any greater than the chance variation between any two patients of the same sex. Another point of interest worthy of further consideration and study may be the much greater variation in the daily dosage of antipsychotic drugs in the female patients compared with the males as noted in both studies.

Notwithstanding the above comments, I believe that such studies on drug-prescription practices in mental handicap institutions serve the important function of increasing the psychiatrists' awareness of the inadequacy of the current practice and motivating them to seek improvement via the emphasis on more rational prescription guidelines, increase of medical input, introducing regular drug review, and alternative treatment approaches. Prescribing psychoactive drugs for the mentally handicapped patients in long-stay institutions requires extra care and consideration, and the dictum to follow is: "When in doubt, don't!" (Kirman, 1975).

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AUTHORS' REPLY: We were interested to read Dr Fan's letter and see that his findings with regard to the prescription of antipyschotic drugs in the mentally handicapped are very similar to our own, despite differences in the ethnic and cultural background of his subjects. We would like to reply to the queries that he has raised.

We chose the use of antipsychotic medication in our study rather than other psychotropic drugs because we believe that antipsychotic drugs are prescribed too readily in mental handicap with insufficient pharmacological indications. Furthermore, the adverse effects of long-term prescription of these drugs are more serious than erroneous administration of alternative psychotropic agents.

We agree that it would have been helpful to look at all patients who received antipsychotic drugs four years before our investigation. However, the logistic task of identifying all patients in the hospital in 1982 and perusing their files was considered too major an exercise and, because of problems arising with patients who had died or who had left hospital in the four years before our study, it was likely that any enquiry of this nature would have been incomplete. We are now undertaking a further study examining the cohort of our 1986 sample to see what drugs they are at present receiving.

Dr Fan is quite right to point out our error in stating that female patients in our study received a significantly higher mean daily dose of chlorpromazine equivalents compared with that of the male patients. We agree that the considerable variation in dosage in the female patients is of interest and shows that there are some female mentally handicapped patients who receive very high doses of antipsychotic drugs. Examination of our data indicates that a group of these patients have a history of frequent disturbed behaviour and/or aggression.

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Buspirone in benzodiazepine withdrawal

SIR: Beeley & Hammersley (*Journal*, November 1990, **157**, 777) comment indignantly that our study of buspirone in benzodiazepine withdrawal (*Journal*, August 1990, **157**, 232–238) was clinically irrelevant and unethical. They castigate us for ignoring the "generally accepted" view that "gradual dosage reduction with appropriate psychological treatment is the best way to manage benzodiazepine withdrawal", and for perpetuating "the search down a blind alley for pharmacological short-cuts".

In Newcastle we have long advocated gradual dosage reduction in benzodiazepine withdrawal, which is individually tailored and combined with psychological support (Ashton, 1987, 1989). We have emphasised the distress that benzodiazepine withdrawal can cause in some patients and have drawn attention to the need for psychological help and for tranquilliser support groups (Ashton, 1984). For the past seven years we have conducted a benzodiazepine withdrawal clinic which operates in close liaison with clinical psychologists and with a tranquilliser advice and support group which we helped to establish. Our general policy has been to involve the patients closely in decisions about their own withdrawal regimes.

After experience with over 200 patients at the clinic (and many more at the support group) it is clear that present methods are not ideal. Although 90% of our patients have achieved and maintained benzodiazepine withdrawal (Ashton, 1987), the clinical course has not always been easy and we have learned that some patients do require additional pharmacological support. For example there is a real risk of suicide in withdrawal and a proportion of patients develop major depression requiring treatment with antidepressants (Ashton, 1987).

Hence we felt that it was (and still is) important to evaluate the effect of pharmacological and other treatments in properly controlled studies of benzodiazepine withdrawal. The case for buspirone was particularly relevant at the time that our study was planned and initiated (1985/86) since there was a strong likelihood that this recently released drug, with proven anxiolytic activity (Feighner *et al*, 1982), would be widely used in benzodiazepine withdrawal, especially in general practice, where studies had shown it was effective in generalised anxiety disorder (Murphy *et al*, 1989). The study received the approval of the Joint Ethics Committee of the University and Newcastle District.

A fixed benzodiazepine withdrawal time of four weeks was chosen because we were anxious to obtain evidence of the utility or otherwise of buspirone in the shortest possible time in the minimum number of patients. Withdrawal regimes which are individually tailored may take over a year to complete (Ashton, 1987) and make it difficult to compare treatments. Furthermore, we had evidence that outcome is not affected by rate of withdrawal (Ashton, 1987) and that fairly rapid withdrawal may sometimes be appropriate (Ashton, 1984). We limited participation in the study to patients taking low to moderate doses of benzodiazepines, and most patients were referred because they had experienced difficulties during previous attempts with "generally accepted" methods of withdrawal. The study itself showed that 11 out of 12 patients in the placebo group were successful in achieving and maintaining withdrawal with the method used (one patient dropped out for reasons not connected with withdrawal).

The patients chose to take part in the trial; those who declined were still offered treatment at the clinic. Participants were given a full explanation of the aims and methods of the study; they were informed that they may or may not receive buspirone, and that the drug may or may not be helpful. There was no reason to anticipate that the drug might exacerbate withdrawal symptoms. It is not true, as Beeley & Hammersley claim, that the regimen took no account of the patients' response to withdrawal. All patients were able to discontinue the trial at any time. Indeed, many who were taking buspirone dropped out because of increasing symptoms or need for further medication, and the incidence of drop-out was one of the criteria used for assessing the effects of the drug. All patients were assessed at frequent intervals by consultant pharmacologists and psychiatrists experienced in benzodiazepine withdrawal. Patients were free to attend a support group or to be referred for psychological treatment if indicated. Those who dropped out were able to continue attending the clinic (and many were later successful in benzodiazepine withdrawal).

We feel that our study was useful in demonstrating that the use of buspirone in benzodiazepine withdrawal was associated with an increased drop-out rate, and possibly more severe symptoms, compared with dosage reduction under placebo. Our study does not perpetuate the use of a pharmacological short-cut; on the contrary, it shows that one pharmacological treatment is a cul-de-sac.

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Anorexia nervosa across cultures

Sir: I was excited to read the letter by Khandelwal & Saxena (*Journal*, November 1990, **157**, 784) who reported that in India anorexia nervosa (AN) is not only rare but is also not associated with any body image distortion and fear of obesity. As these have been regarded as 'core psychopathology' of AN and are of diagnostic importance in the West, their patients do not fulfil DSM–III–R criteria and are placed in the residual category of 'eating disorders not otherwise specified'. However, they were noted to be amenorrheic and rigidly maintain a low body weight just as Western AN patients do. These findings echoed my study of Chinese AN patients in Hong Kong, where a clear distorted body image or an intense fear of obesity is lacking (Lee *et al*, 1989).

AN has often been described as a culture-bound syndrome. While there is more evidence on the greater prevalence of AN in Western than non-Western countries, much less information is available as to whether its clinical patterns also differ across cultures. I believe that they do, and would suggest that more detailed case histories of these Indian patients should be provided for cross-cultural comparison. Besides, although the three Asian adolescents reported by Bhadrinath (1990) had "considerable fears of getting fat and body image disturbances" and met DSM–III–R criteria, it must be noted that they all came to the UK as a baby or as a child and thus grew up with Western notions of slimness and dietary preoccupations. This phenomenon of acculturation has been studied by Mumford & Whitehouse (1988), who found bulimia nervosa to be less common among white girls than second-generation young British Asians, who are quick to learn fashionable Western styles of reacting to stress.

Diagnostic criteria for AN need to be appreciated in the context of the attitudes to food, eating and body shape of a particular culture. It is vital that psychiatrists from other non-Western cultures do not apply DSM–III–R rigidly, and report on any atypicality in the AN patients they see. This will contribute to important cross-cultural data and a more culture-free understanding of abnormal female fasting, which has a long history, and in Western countries has happened to be defined in 'atheoretical' diagnostic manuals as the AN which most of us recognise today. There is really a difference between 'culture-bound' and 'concept-bound' entities.

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MUMFORD, D. B. & WHITEHOUSE, A. M. (1988) Increased prevalence of bulimia nervosa among British schoolgirls *British Medical Journal*, **297**, 718.

SIR: The recent surge of interest by professionals in eating disorders among Asian people is long overdue. Bhadrinath's highlighting of the condition (*Journal*, April 1990, **156**, 565–568) described an important phenomenon which is probably far more common than has so far been reported. Certainly the EAT score data of Dolan *et al* (*Journal*, October 1990, **157**, 523–528) would suggest this to be so.

From our own recent experience in treating a 13-year-old Asian girl, who fulfilled DSM–III–R criteria for anorexia nervosa, there was evidence to suggest that the condition was precipitated and maintained by features associated with problems relating to cultural conflict. The increased sensitivity to body appearance, common during adolescence, was heightened by the cultural conflict she experienced while negotiating the task of being Asian in the United Kingdom.

One of her main reasons for not eating was the desire to look and dress like 'white models' in fashion magazines and in this way feel more part of western culture at the same time as differentiating herself from that of her parents.

Further work is needed to explore the epidemiology of eating disorders among Asians and the hypothesis that the pursuit of 'westernness' contributes to the aetiology needs to be investigated.

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SIR: I read with interest the comments of Khandelwal & Saxena (*Journal*, November 1990, **157**, 784) in response to Badrinath's article (*Journal*, April 1990, **156**, 565–568).

The variance of anorexia nervosa described by Drs Khandelwal & Saxena highlights an important deficit in the diagnostic practice which fails to distinguish genuine anorexia nervosa from the atypical one. The young female described by them with decreased appetite, excessive weight loss and amenorrhoea, but no clear body image disturbance or fear of becoming fat, may have something else but not 'genuine' anorexia nervosa. Such differentiation becomes more important when we try to understand this disorder in the cross-cultural context.

The concept of anorexia nervosa suggests that the weight loss, emaciation and other characteristics are secondary to a relentless pursuit of thinness which appears to be the primary preoccupation in these patients. Mixing the concepts of genuine anorexia nervosa and the one which is not characterised by this preoccupation about thinness defeats the very purpose of understanding it conceptually and culturally.

When Simmonds (1914), a pathologist, reported a destructive lesion in the pituitary gland, every case of malnutrition was explained as caused by some endocrine pathology (Brusch, 1975). Over the next two decades genuine anorexia nervosa was filtered away from Simmonds disease. Now, when the understanding of its psychopathogenesis and evolution is becoming clearer, it will be a backward step not to view anorexia nervosa and atypical eating disorder as distinct disorders.

The syndrome seen rarely in the Indian subcontinent is the one characterised by body image disturbance and preoccupation with weight reduction. In that sense anorexia nervosa may be a culture-bound syndrome of the West, influenced by the western cultural norms and practices.

Whereas traditionally in the Indian culture fullness of the body has been regarded as a sign of a well nourished, healthy, affluent and beautiful lady, western beliefs, values, perception and behaviour regard the pursuit of thinness as a perfectly logical concept of beauty. Thus it will not be wrong to suggest that the western socioculture itself dictates this particular behaviour.

Until there is significant weight loss and emaciation, psychiatrists in the West fail to recognise this impairment in judgement and deficient insight about her condition in an anorexia nervosa patient. That is probably also the reason why the classificatory system, which is entirely 'western', places more emphasis on measuring physical parameters for making a diagnosis of anorexia nervosa, than on recognising the overvalued idea or even a delusion of being obese as primary psychopathology.

Solitary cases of this disorder seen in the children of Asian migrants to the West only substantiates the fact that it is the western culture which influences this disorder. As the Indian subcontinent becomes more 'westernised' and adopts the value systems of the West, it will be surprising if this culture-bound syndrome of the West does not percolate to the East.

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Anorexia nervosa in the elderly

SIR: We read with interest the report of Gowers & Crisp of an 80-year-old woman with anorexia nervosa (*Journal*, November 1990, **157**, 754–757). Although anorexia nervosa is considered to be rare in the elderly it is perhaps unsurprising that such cases exist given the significant incidence in young people and the high rate of chronicity associated with the disorder.

Among schoolgirls, Crisp has previously shown an incidence of one severe case in every 200 over the age of 11 years, rising to one in every 100 over the age of 16 years (Crisp *et al*, 1976). This may still be an underestimate given the rate of body-shape dissatisfaction among British comprehensive schoolgirls found by Salmons *et al* (1988). In their survey some 25% to 30% of girls aged 16 to 18 years admitted to being only rarely satisfied with their bodyweight, and 'usually' or 'always' terrified of gaining weight.

A multitude of studies on the outcome of anorexia nervosa have demonstrated a high rate of chronicity. Hsu (1980) and Schwartz & Thompson (1981), reviewing the more rigorous studies which had appeared over the preceding 15 years, found a general recognition that some 50% of cases showed continued abnormal eating behaviour at follow-up, which extended in some surveys up to 35 years after the time of first diagnosis. Even if this is an overestimate, the implication is that many people who suffer from anorexia nervosa in their youth maintain their abnormal eating attitudes throughout their lives, and it is perhaps surprising that anorexia nervosa is not described more frequently in the elderly.

We have recently treated a 73-year-old woman with features similar to the woman described by Gowers & Crisp (Cosford & Arnold, 1990). She suffered an episode of anorexia nervosa following a bereavement at the age of 23 years. This was characterised by marked weight loss, extreme behaviour to avoid food intake, an expressed fear of gaining weight, and secondary amenorrhoea. She recovered after nine months of in-patient treatment and subsequently maintained an adequate weight for some 50 years afterwards. She recently suffered a relapse, with severe weight loss, a distorted body image and a fear of becoming fat. Her eating behaviour again became markedly abnormal, and multiple investigations failed to reveal a physical cause for her weight loss. She responded to a strict dietary regime and was discharged five months after admission having regained her former weight, which she has subsequently maintained during out-patient follow-up.

We would suggest that anorexia nervosa is probably underdiagnosed in the elderly, and would support the assertion that it should be included in the differential diagnosis of unexplained weight loss in this age group.

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Psychological sequelae of torture

SIR: I was grateful to read your annotation on psychological sequelae of torture by Turner & Gorst-Unsworth (*Journal*, October 1990, **157**, 475–480).

Torture has been a widespread experience in the 20th century. Most probably there has never been a time when institutionalised torture has been so widely inflicted on large masses of people in all continents. After the military coup of 1980 in Turkey, I witnessed large numbers of victims of torture in Metris Military Prison, Istanbul, where I was imprisoned for one year (1982–1983). I would agree with the authors that torture has a wide variety of psychological effects on the victims, their families and friends. But psychological sequelae of torture cannot be limited to them, but should be extended to the large group of torturers who have been especially trained to torture.

The situation where individuals first have been forced, then slowly taught, to obey and then to enjoy human suffering, and to become professionally trained systematic torturers must be considered. Having had the opportunity to observe torturers, one cannot help feeling for those who have most probably in their turn been psychologically and physically abused. In Turkey, many turturers warrant diagnoses of psychiatric syndromes which have never been diagnosed or treated. Suicide rate, deliberate self-harm, alcohol dependency and possibly other drug misuse appears to be much higher in those individuals trained to be involved in torture. Different forms of psychotic episodes are commonplace and homicide rates among torturers are much higher than in the general population.

Torture has wide implications upon the whole society where its practice takes place. The society as a whole gets enmeshed into the idea of its existence, and fear and degradation is extended to all aspects of life. Now in Turkey torture has become a major theme in short stories, poetry, films, pictures and songs. In the last ten years there have been hundreds of poetry books, short stories, paintings and films on the tortured, the torturers and their circumstances. It has become part of the language and culture and almost a way of communicating. Its existence transcends all boundaries and makes itself felt in all aspects of life.

In my out-patient clinic at the Charing Cross Hospital, Turkish immigrants who have never experienced torture come with stories of ill-treatment as their psychological complaints. Both neuroses and psychoses in these people are flavoured with stories of torture, sufferings and horror. The idea of torture, even if they know little about it, has become an expression of their persecutions, anxieties, racist and sexist experiences. It is a component of their guilt, self-pity and hopelessness. The individual and the whole society has been marred by the psychological effects of torture.

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British and Australian depression revisited

SIR: Two recent long-term outcome studies of depression, from Sydney (Kiloh *et al*, 1988; Andrews *et al*, 1990) and London (Lee & Murray, 1988; Duggan *et al*, 1990), have shown similar results. The initial diagnosis often heralded a very poor long-term outcome, and personality disturbance was one part of the explanation for this. However, there are important differences between the Sydney and London findings which we wish to highlight.

The first of these concerns the neuroticism (N) subscale of the Eysenck Personality Inventory (EPI). The EPI N now has an excellent pedigree as a predictor of outcome in depression, but Professor Andrews *et al* found its predictive power to be confined to their subgroup of 'neurotic' depressives. This may encourage readers to re-identify raised N with a diagnosis of 'neurotic' depression, which would be unwise. In both series, N scores do not differ between 'neurotic' and 'endogenous' subtypes, so that whatever separates the 'neurotic' from the 'endogenous' depressive, it is not the degree of neuroticism. In London, unlike Sydney, we found that EPI N predicted chronicity particularly in the 'endogenous' (melancholic) subgroup. We therefore propose that the relationship between high N scores, diagnostic subgroup, and outcome should remain open to further investigation.

A second difference concerns the influence of depressed mood on the assessment of personality Andrews *et al* apologise that their patients were assessed when they still had symptoms, and argue that 'recovered' personality has the more significant influence on outcome. We were able to compare the effect of personality estimated when ill and again on recovery and found that the former was the more important. This is in agreement with findings from cognitive psychology, where significant differences in dysfunctional attitudes between depressives and normal controls are found only in the presence of depressed mood. Thus it may be that self-ratings of personality are more valuable predictors when an individual is in a state of depression rather than on recovery, and that the changes produced by the depression are precisely those which offer most information for the future.

Our third point of difference concerns diagnosis. Professor Andrews *et al* claim to show that 'endogeneity' in depression has at most only a trivial effect on long-term outcome. However, this conclusion depends heavily on their definition of 'endogenous' depression – there are at least 18 different definitions and the degree of overlap between them is only partial. We have compared the prognostic value of several of these in our Maudsley series and have found (a) that differing definitions of 'endogenous' depression include different populations and (b) DSM–III melancholia emerges as the best predictor of poor long-term outcome (Duggan *et al*, 1991). Thus, it may be premature to dismiss the importance of the endogenous/nonendogenous distinction, as Professor Andrews *et al* have done.

Finally, the Sydney group continue to misrepresent our earlier papers. In London, 'endogenous' scores on Kendell's continuum predicted a much poorer outcome. Despite several appeals, it was the swift hare, not the slow tortoise, that lost Aesop's race.

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Urinary chromatographic profiles in psychiatric diseases

Sir: In 1980 Trygstad *et al* reported that patients with a variety of psychiatric diseases could be distinguished from each other and from normal controls by the pattern of peptides excreted in urine as studied by chromatography. Specifically, it was claimed that when urine was precipitated by ethanol saturated with benzoic acid, centrifuged at 4000 *g* for 10 minutes and the precipitate washed with ethanol, dissolved in ammonium bicarbonate and applied to a Sephadex G-25 column, the following distinctions in ultraviolet absorption profile could be made. Firstly, patients with unipolar and bipolar depression had patterns which differed from normal subjects and patients with neurotic depression, and these profiles were normalised by tricyclic antidepressant medication. Secondly, patients with hebephrenic or paranoid schizophrenia differed from normal subjects in demonstrating one or other of two chromatographic profiles characterised by an excess or deficit of peaks of absorption seen at elution volumes between 1200 ml to 1400 ml. Thirdly, patients with autism had excretion profiles which were similar to those seen in schizophrenia, but which could be distinguished from these latter on further (unspecified) fractionation steps. Fourthly, patients with hyperkinetic syndrome (minimal brain dysfunction) could be shown to have a distinct, but variable, pattern of urinary peptide excretion which returned to normal with amphetamine treatment.

These and related claims were made in a series of papers (e.g. Reichelt *et al*, 1981, 1985, 1986) by the same group of workers.

To assess these claims we have conducted an investigation of urine samples from five neuroleptic-free patients with schizophrenia (DSM–III criteria) and four age- and sex-matched normal subjects, using Sephadex G-25 and Biogel P-2 chromatography with techniques as described by the Norwegian group and a series of modifications (Gilroy *et al*, 1990). In the course of our investigation a number of difficulties with these techniques became apparent. In communication with the Norwegian workers, we have attempted to clarify the nature of the difficulties, and to further specify the precise procedures adopted. Details of these technical problems are given in our paper (Gilroy *et al*, 1990).

Our findings differ from those of the Norwegian group. We observe urinary chromatographic profiles which do not closely resemble those which they have reported. We found substantial differences in excretion profile between men and women on Sephadex G-25, but no significant differences (in our small sample) between patients with schizophrenia and controls on either Sephadex G-25 or Biogel P-2. Our findings give no support to the view that the patients with schizophrenia can be readily distinguished from normal subjects by an analysis of the chromatographic profile of peptide excretion in urine.

Although we have not studied patients with unipolar and bipolar depression, autism or the hyperkinetic syndrome we consider that the uncertainties concerning the precise methods adopted by the Norwegian workers and the technical difficulties revealed in the course of our investigation cast their conclusions concerning the role of peptides in these conditions in doubt.

The methods used are complex, with many possible sources of variation, and we suggest that a more rigorous and quantitative approach than that so far adopted by this group of workers is required before these findings can be regarded as reflecting on the nature of the disease processes in question.

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Diazepam abreaction

SIR: Recently, Ellis (1990) made some interesting observations about the role of diazepam and other sedative drugs in abreaction interviews. However, this is not the first time such a practice has been adopted. In fact, this practice has been in routine use for more than 15 years in preference to 'amytal test' at the Department of Psychiatry, All India Institute of Medical Sciences, New Delhi, where some of us trained as psychiatrists in the early 1980s. In India, conversion hysteria is a very common clinical diagnosis not only in psychiatric out-patient clinics but in general practice and medical out-patients as well. Intravenous diazepam abreaction interview is generally much safer as compared with the 'amytal test' and can be useful in primary care settings where facilities for intubation and resuscitation are not very good. It is in this setting that a doctor in India encounters numerous cases of conversion hysteria. The use of diazepam abreaction is so common there that one does not consider it to be a rarity worth publishing. We have, incidentally, mentioned this clinical use of diazepam while discussing case histories of patients with multiple personality disorder (Adityanjee *et al*, 1989).

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Racial stereotypes

SIR: The paper by Lewis *et al* (*Journal*, September 1990, **157**, 410–415), is to be commended as an attempt to elucidate British racial stereotypes that may influence diagnostic practice. However, I do not think the study addresses the issue of racism in psychiatry – at least not very fully – and the title of the paper ("Are British psychiatricts racist?") is a misnomer.

In their report on the influence of racial stereotyping on diagnosis, the authors state that their findings refute the claim that British psychiatrists tend to over-diagnose schizophrenia among Afro-Caribbeans. I do not think that such a conclusion can be drawn from their study as reported. The use of case vignettes is a useful tool in this type of research in spite of the obvious drawback (referred to by the authors) that the attitude to a hypothetical case may not reflect actual practice; and the method has yielded interesting results in an earlier American study (Loring & Powell, 1988) quoted by the authors. However, in using a vignette, great care must be taken to ensure that it is not itself biased with respect to the factors being studied or else that a sufficient number of vignettes are used to control for such bias. (In the American study the researchers controlled for this sort of racial/gender bias by using two vignettes reflecting real cases of a black male and a white female.) Since Drs Lewis *et al* used only one case vignette (which was varied four-ways by altering gender and race), their methodology should have ensured that it did not carry elements within it that raised images of race or gender (apart from direct designation of race and gender). Unfortunately, the authors do not tell us how their vignette was derived. Did it reflect an actual case and if so what was the original gender and race? How did the researchers ensure that the vignette they used did not contain a racial or gender bias (apart from race and gender stated directly)? For example, did they test the vignette devoid of racial and gender categorisation in pilot studies?

I suggest that in reading the case vignette given in their report the image (via a sort of stereotyping) that may develop in the mind of the psychiatrist is of a *black* person before the point is reached (fairly late in the description) when the race of the person is mentioned. I think that the references to religious interest, to the father being a British Rail ticket clerk and to the smoking of cannabis *taken together* may ensure this happening. Therefore I suggest that if, as seems likely, the case vignette used by the researchers gave an image of a black person, this may well persist in at least some instances even when the person is identified as being white. Hence, some (many?) of the 'white' people diagnosed as schizophrenic may have been visualised (as it were) as 'black'. All this may seem far-fetched to the naive reader/researcher. I suggest that neither racism in psychiatry nor the influence of stereotypes in psychiatric diagnosis is simple or straightforward (Fernando, 1988, pp. 44–49). Research in these fields must be handled with sophistication.

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Stress and puerperal psychosis

SIR: The suggestion by Brockington *et al* (*Journal*, September 1990, **157**, 331–334) that, unlike post-natal depression, pre-natal depression is strongly associated with social stress, particularly life events in their investigation, appears to be supported by a study that we have conducted recently (Kitamura *et al*, in preparation).

Of 120 consecutive women recruited from among those attending an antenatal clinic in the obstetrics department of a general hospital in Japan, 19 (16%) were identified as showing onset of affective disorders during their period of pregnancy according to the Research Diagnostic Criteria (Spitzer *et al*, 1978), mainly major depressive disorder ($n = 13$). Interviews were conducted with the Schedule for the Affective Disorders and Schizophrenia (Spitzer & Endicott, 1978). As compared with women without onset of affective disorders (controls), the depressed women were characterised by (a) either first pregnancy or first delivery with past termination of pregnancy (28% v. 5%), (b) early loss of either parent by death (21% v. 5%), (c) low degree of paternal care and maternal overprotection during childhood (26% v. 8%), (d) high scores on the neuroticism (11.8 [s.d. 4.3] v. 8.7 [s.d. 4.6]) and psychoticism (4.4 [s.d. 2.1] v. 3.1 [s.d. 1.9]), subscales of the Eysenck Personality Questionnaire, (e) living in a flat with the expectation of either staying there after the childbirth or that accommodation would become crowded (29% v. 6%), and (f) negative response to the news of the pregnancy by the husband, with a low degree of intimacy (63% v. 13%). The effects of these factors were additive, since the probability of developing the affective disorders was highly correlated with the number of these factors.

These findings and those of Professor Brockington *et al* (1990) strongly indicate that pre-natal depression is mediated through a variety of psychosocial stressors. It seems, therefore, that pre-natal depression is a long-neglected area, warranting further investigation.

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Erotomania in relation to childbirth

SIR: Murray *et al* (*Journal*, June 1990, **156**, 896) do not discuss the possible role of alcohol in the illness of their patient. Organic factors are well known in the aetiology of this disorder, as described in a number of the references they list. Their patient is described as "never a heavy drinker, she drank two cans of beer most nights in the six months before referral". A statement like this by a 40-year-old mother of two children should have made one press harder about the history of alcohol consumption. For instance, what does "never a heavy drinker" mean? Did she drink spirits? What beer did she drink – some beers are approximately equivalent to six units per can? Is there any independent confirmation of the alcohol history? Is there any question of other drug abuse? In this connection one is bound to note that there were financial problems and that her husband was anxious and irritable and one wonders whether he might have been drinking as well.

Symptoms caused by alcohol would be expected to subside within a matter of weeks in most cases. If alcohol had been considered then she would have been kept in hospital for some weeks for diagnostic purposes before beginning drug treatment, and only if symptoms persisted would other diagnostic possibilities have been considered. One would therefore like to know how long after admission was the trifluoroperazine started, and how soon the resolution of symptoms began.

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Effect of beliefs on grief

SIR: Kavanagh (*Journal*, September 1990, **157**, 373–383), in his otherwise stimulating review of adult grief reactions, almost totally ignores the effect of an individual's belief or not in an 'after life'. Such beliefs can have a significant impact on the attitude of the recently bereaved person to the loss. Dr Kavanagh clearly identifies the possible conflicts between belief and actual experience: "A continuing problem for many people is the challenge that the death can pose to central attitudes by which we maintain goal-directed behaviour . . . beliefs about the meaningfulness and fairness of existence . . . belief in a divine being . . . may also come under threat". However, Dr Kavanagh fails to incorporate an understanding of the benefits such beliefs may give to the sufferer. In the cognitive-behavioural interpretation that he suggests, "normative issues are discussed and irrational guilt is minimised".

Whose baseline is taken in deciding 'normative' and 'irrational'? A firmly held Christian belief in an afterlife of Heaven and Hell would be interpreted by many mental health workers as 'abnormal' and 'irrational'. Yet, to challenge and attempt to deny the sufferer's belief system would, I suggest, be to exacerbate their already significant and normal distress.

Any intervention for grief must make allowance for the philosophical or religious attitudes of the bereaved towards the meaning of life and death. This will help to achieve Dr Kavanagh's laudable aim "to maximise survivors' achievements and minimise the pain they suffer to gain them."

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SLE and multi-infarct dementia

SIR: Green (*Journal*, November 1989, **155**, 707–711) published the account of a 54-year-old woman with abnormal involuntary movements, who, over the years, had been given diagnoses of hysterical conversion syndrome and bipolar affective disorder. It transpired that a single diagnosis of systemic lupus erythematosus (SLE) could account for all these features, based on the evidence of selective micro-infarcts in the frontal and temporal lobes and serum autoantibodies to DNA.

We describe a second case of SLE presenting with protean psychiatric symptoms, again with discrete microinfarcts, this time in the frontal and temporal regions on nuclear magnetic resonance (NMR) imaging.

Case report: A 70-year-old Caucasian woman was transferred to Mossley Hill Hospital with a subcortical dementia-like picture. She had a deadpan expression, was uncommunicative and glided silently about the ward, occasionally with tears streaming down her face. Sometimes she would vary her behaviour by answering questions monosyllabically, or lying down on the floor or attacking other patients.

lowing the drowning of a grandchild, she presented to atric care in the USA in 1984 with a 'major depressive er with psychotic features'. She responded to tri- and neuroleptics. This was followed by a relapse of pression with a 'paranoid psychosis' in 1985 after the of another grandchild. She was treated with electro- lsive therapy (ECT). Having returned to England, she d a further depressive illness in 1988 which featured ic delusions, evidence of short-term memory loss, haviour such as throwing herself on the floor or lying chairs. She responded slowly to tricyclics and anti- sants. Her face was mask-like and her responses were Her recovery was limited and a presumptive diagnosis ti-infarct dementia was made based on the cognitive s of disorientation, short-term and long-term ry loss and dyscalculia together with the NMR report 5 quoted above.

rum autoantibody screen performed in 1990, how- evealed markedly abnormal homogenous autoanti- suggestive of SLE. This, together with the NMR of microinfarcts in 1985, suggests that a single diag- f SLE would account for this woman's six-year psy- c career. It is of note that she had no family history of nor psychiatric disorder, and that her past medical showed no manifestation of SLE in any other bodily . Her pre-morbid personality was that of a religious, ng and friendly woman, used to public speaking and nate about her garden

There are echoes between this case and the earlier one described by Dr Green – both are manifestations of solely cerebral SLE diagnosed after a career of affective illness. In the early stages, carers and case notes of both cases refer to 'histrionic', 'hysterical', 'negativistic' or 'paradoxical' behaviour. The later stage of both cases appears to be characterised by movement disorders, one being a focal athetosis and the other a strange gliding gait and Parkinsonian facies.

If SLE can be seen twice in the psychiatric catchment area of Liverpool, it may follow that there is a much larger number of cases elsewhere, remaining as yet undiagnosed. If the diagnosis can be made and a series established then perhaps we will be that nearer a treatment for this most disabling condition.

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A HUNDRED YEARS AGO

n-restraint question

ere under the impression that the discussion en Dr Yellowlees and Dr Alex Robertson had sted itself in our last number. Each physician lly and freely expressed his views on a subject ard to which they honestly hold different ns. To continue the discussion would, we be little more than a repetition of the same ents, if not the same words, without adding any rce to the arguments employed by these able tants. Dr Robertson, however, wished to make istakably clear that he regards "locked gloves" form of mechanical restraint. As he places in the ategory "side arm dresses" and the "protection bed", and as Dr Yellowlees recommends their use in exceptional cases, Dr Robertson maintains that he was not in error referring to "the considerable use of mechanical restraint" advocated by him. Another statement Dr Robertson wishes to make, which is, that although he has been connected with an asylum which during the last five years has not had a larger number of patients than 125, it was, during many years previously, licensed for 248 patients, a large proportion of whom were dangerous, both in respect of suicide and homicide.

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British Journal of Psychiatry (1991), **158**, 293–295

Books Reconsidered

La Schizophrénie: E. Minkowski

Eugene Minkowski was born in St Petersburg, educated in Warsaw and obtained his medical education in Munich. He moved to France in 1915, served in the medical corps attached to the French army throughout that war and was awarded several medals for bravery. He then settled down in Paris, where he remained as a practising psychiatrist until he died in 1972. He was never a professor, nor even a consultant in a prestigious psychiatric hospital, but he was able to command respect from all who knew him.

La Schizophrénie, subtitled *Psychopathologie des Schizoides et des Schizophrènes,* was first published in 1927 and a revised edition appeared in 1953. At the time of the original edition Bleuler's book *Dementia Praecox oder Die Gruppe Schizophrenien* (1911) had not been translated into French and the Kraepelinian-Bleulerian notion of schizophrenia, encompassing virtually all forms of non-organic, non-affective psychosis, was not accepted by French psychiatrists. They subscribed to a more manifold classification of psychosis in which a variety of paranoid states were distinguished from hebephrenia or catatonia. It was essentially a pre-Kraepelinian classification which has persisted in attenuated form to this day.

Minkowski's aim in writing the book was to convince his compatriots that Bleuler was right in his overall scheme. However, Minkowski's intention was not merely to provide a synopsis of Bleuler's ideas for those who could not or would not read German. He wanted to put across his own quite specific and unique views about the essential psychopathology of schizophrenia, while acknowledging that "despite our divergence of opinion I am still a pupil of Bleuler. I walk in the furrow which he ploughed".

It is the nature of Minkowski's divergence from Bleuler which I want to address in this article. Until I translated one of the chapters of this book (Cutting & Shepherd, 1987), Minkowski was virtually unknown to English-speaking psychiatrists. I have rarely seen any reference to his numerous publications. Laing (1959) referred to him in his *Divided Self,* and he is sometimes regarded as an 'existential psychiatrist'. However, Laing was much more influenced by Binswanger, who, in my view, was more abstruse and quite wrong in his claims about the condition.

What was then Minkowski's contribution to the psychopathology of schizophrenia and why should we bother about it today? After all, what with magnetic resonance imaging, positron emission tomography, single photon emission computerised tomography, etc, isn't the clue to the nature of schizophrenia just around the corner?

Minkowski's claim on our attention is simply that, in my opinion, he provides the most comprehensive psychological account of schizophrenia ever written. Moreover, although his scheme lacks any biological foundation, his observations are so accurate and profound that not only can they be immediately accommodated within current neuropsychological models of schizophrenia (see Cutting, 1990), but they also illuminate and help to confirm neuropsychological theories of the mind which were formulated without reference to psychiatric disorders at all (e.g. Kosslyn, 1987). He appreciated how sterile the psychological theories of mind of his time were – associationism, psychoanalysis, Gestalt psychology. More striking, he had the ability to leap-frog over the equally bankrupt theoretical schools which came later – behaviourism, cognitive psychology – and formulate a theory of mind which is in complete accord with current neuropsychological thinking. He was, in short, ahead of his time, but, more than this, he was the first person, to my knowledge, to appreciate that a correct understanding of the nature of schizophrenia can illuminate the nature of the normal mind. All commentators, before and since, have adopted a one-way approach, trying to explain schizophrenia in terms of some theory of normal functioning (which has invariably turned out to be false). All, that is, except Jaspers, who at least had the sense to appreciate that it was 'un-understandable' according to any psychological theory or biological mechanism that he knew of. Minkowski saw schizophrenia as allowing insights into normality just as much as normality could illuminate the schizophrenic mind.

First, what did Minkowski reject of Bleuler's scheme? He disagreed with Bleuler on two points. He did not believe that a loosening of associations was an adequate explanation of the entire clinical picture, and he did not consider that emotional complexes had any specific bearing on the development of particular symptoms. It is not entirely clear why he

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1953 (2nd edn) Paris. Desclée de Brouwer rejected the 'loosening of associations' theory, but it is probable that he simply saw his own theory as a better alternative. As we have seen, he was keen to promote the Bleulerian view of schizophrenia in France, referred to Bleuler at several points as his '*maître*', and therefore probably did not want to discredit him too much. As for the influence of Freud and Jung on Bleuler's views, Minkowski is more definite. Although generally sympathetic to psychoanalytic views of normal human development, he could see no role for them in causing or even moulding any aspect of schizophrenia.

Minkowski's second acknowledged '*maître*' was Henri Bergson, the French philosopher whose most well known legacy is probably the concept of a 'stream of consciousness'. A cursory acquaintance with Bergson's views on the nature of the mind is essential to understand Minkowski's ideas, because, if there was any theory of the normal mind which he tried to apply to schizophrenia, it was Bergson's.

Bergson believed that there were two forces which determined a normal person's mental life. One was 'intellect', the other 'instinct'. These two forces were diametrically opposed to one another, in that each treated the world in completely different ways.

'Intellect' was the power of seeing things as separate from one another, frozen in time and separate in space – what he called the 'cinematographic' representation of the world. Bergson was writing at the dawn of the silent film era, and it was not unnatural that he should have used this analogy. Another analogy for the property of 'intellect', used by Bertrand Russell (1946) in his commentary on Bergson, is of a carver of chicken. The 'intellect' carves up the chicken, but with "the peculiarity of imagining that the chicken always was the separate pieces into which the carving-knife divides it".

It is much harder to understand what Bergson meant by 'instinct'. His analogies belong to the realm of poetry: it is a "cavalry charge", it is a "shell which bursts into parts", it is "life itself". Somehow, 'instinct' represents the fluidity of things across time, blurring memory and perception. It also encompasses intuition or common sense.

The difficulty one has in grasping the essence of this world-view should not detract from its correctness, according to Bergson, because he wrote into it the proviso that 'intellect' is inherently incapable of understanding the nature of 'instinct'! Minkowski certainly thought that he understood it and thought that the psychopathology of schizophrenia was a living illustration of its validity. Consider this account by one of his schizophrenic patients:

> "Everything is immobile around me. Things present themselves in isolation on their own, without evoking any response in me. Some things which ought to bring back a memory, or even conjure up a thought or give rise to a picture, remain isolated. They seem to be understood rather than experienced. It is as if a pantomine were going on around me, one which I cannot take part in. There is nothing wrong with my judgement but I seem to lack any instinctive feel for life. I can't change from one emotion to another; and how can you live like that? I've lost contact with all sorts of things. The value and complexity of things no longer exists. There's no link between them and me. Everything seems frozen around me. I have even less scope for manoeuvre with respect to the future than I have about the present or the past. There is, inside me, a sort of routine which makes me quite incapable of imagining the future. Any creative ability is completely abolished. I can only see the future as a repetition of the past."

Minkowski saw overwhelming evidence in this account for a deficiency in one of Bergson's two mental forces – 'instinct' – with, at the same time, preservation of the other – 'intelligence'. He called this deficiency "lack of vital contact with reality" ("*perte du contact vital avec la réalité*"). Certainly, the words used by his patient – "I seem to lack any instinctive feel for life", "creative ability ... abolished", "things ... understood rather than experienced" – fit neatly into Bergson's scheme.

The intellectual activities of the mind were not just preserved, according to Minkowski. Bereft of all those "factors relating to instinct", there was now a "compensatory hypertrophy of everything which pertained to intellect". So, for example, there could appear what Minkowski referred to as "morbid rationalisations and preoccupations with geometry" ("*rationalisme et géométrisme morbides*"). Consider these examples:

> "An obsession with pockets made its appearance. He wanted to know what difference there was between putting one's hands straight into a normal jacket pocket and putting them into the sloping pockets of an overcoat. ... He also had the habit of standing in front of a mirror, legs together, trying to place his body symmetrically to achieve, as he said, "an absolutely perfect position."

> "During his military service he had once been given an injection. The idea had then grown on him that a piece of cotton wool had entered his body, along with the injected fluid. ... The obsession grew and grew. It was no longer just cotton wool that had been inserted, it was the metal from the needle as well, the glass from the syringe; "each organ in my body was systematically affected, until my brain was involved".

> "I was tormented by the vaults in churches. I could not accept that all that weight could be supported by ribs, pillars and a keystone. I could not understand why it did not fall down. I could not see why the cement in the free stones did not crumble, because it must be a particularly vulnerable pressure point. I concluded that houses stayed up only through some terrestrial attraction. I came to doubt my own senses."

Minkowski regarded such preoccupations as evidence of an enhancement in these patients of all that Bergson's notion of 'intellect' stood for.

His most ambitious proposal was to list dichotomous properties of the mind or qualities of the world which the mind surveys, in each of which pair one element was 'atrophied' and the other 'hypertrophied'

Atrophied	*Hypertrophied*
Life	Planning
Instinct	Brain
Feeling	Thought
Faculty of penetration which synthesises	Analysis of details
Impressions	Proof
Movement	Immobility
Events and people	Objects
Realisation	Representation
Time	Space
Succession	Extension
End	Means

There are other elements to his theory which can only be touched on here. One is his discussion of autistic thinking. He notes that Bleuler revised his own ideas on this in 1921 by proposing that the term '*dereirendes Denken*' ("thinking which takes no account of reality or deviates from it") should replace '*autistisches Denken*'. This is closer to his own view of schizophrenia as primarily a mind turned away from reality rather than a mind primarily turned in on itself. Another profound insight is his introduction of the concept of 'a pragmatic deficit' ('*démence pragmatique*', '*déficit pragmatique*') as central to schizophrenia. Not only does it show that he appreciates the difference between the intellectual deterioration of, say, Alzheimer's disease and the mental deterioration in schizophrenia, but his very use of the word pragmatic antedates by decades the 'discovery' of pragmatic language by linguists in the 1950s.

> "A schizophrenic knows the date but this knowledge has no precise meaning for him. He can't use it in a way which is appropriate to the circumstances. The pragmatic factor of things is affected from a very early stage."

The relationship between schizoid personality and schizophrenia itself also concerned him. He regards the former as a *forme fruste* of the latter, in which the atrophy of 'instinct' and hypertrophy of 'intellect' are slight, thus allowing a relatively normal life.

I hope I have given a flavour of Minkowski's ideas. The curious thing to me is why he has been neglected, certainly in the English-speaking world. Perhaps it is a consequence of the general low esteem in which psychopathology has been held since the last war. Perhaps it is the suspicion aroused by the irrationalism and poetic imagery of Bergson's philosophy which inspired him. Most likely of all, it is the fact that it is in French, and the average professional in Britain now no longer reads foreign languages.

What of the current value of Minkowski's scheme, outlined earlier? For me, it is correct at three levels.

First, it provides a convincing account of the world-view of a schizophrenic. It certainly accords well with my own records of their experience, and illuminates many observations that I had thought trivial at the time. For example, one of my patients had spent all his days for several years before his illness was diagnosed and treated cutting out geometrical shapes. After three weeks on neuroleptics this behaviour completely disappeared. This was clearly an example of a morbid preoccupation with geometry.

Secondly, Minkowski's scheme of atrophy of certain mental functions and a compensatory hypertrophy of contrasting functions is remarkably similar to the particular pattern of deficient and overactive mental systems which I believe is the critical psychological substrate to the condition, explicable in terms of right-hemisphere dysfunction (see Cutting, 1990). If we consider that it was not until the 1940s that any specific functions at all were attributed to the right hemisphere, Minkowski's prescience on this matter is all the more astounding.

Finally, his and Bergson's notion of the normal state of affairs as consisting of two opposing forces – irrational and rational – is in my view quite correct. This, of course, is also a core concept in psychoanalytic theory. However, the particular interaction which Minkowski and Bergson proposed as existing *between* the rational and irrational parts of the mind is, in my opinion, much superior to that formulated by Freud. The rational mind, in the former scheme, has *no* way of knowing the workings of the irrational part. No amount of dream interpretation, analysis of slips of the tongue or years on the couch will uncover its secrets. It may set up hypotheses, but these will, except by chance, be specious.

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British Journal of Psychiatry (1991), **158**, 296–305

Book Reviews

Editor: Sidney Crown

Seasonal Affective Disorder. Edited by C. THOMPSON and T. SILVERSTONE. London: CNS Publishers. 1989. 278 pp. £19.80.

This is a stimulating and topical book. Here are groups of patients, predominantly women, who each spring or autumn become depressed, gain weight or have a craving for sweet things. In these latter respects they differ from classical endogenous depressives who also have a spring peak incidence. Nosologically, seasonal affective disorder (SAD) does not conform to existing classifications – endogenous/reactive, psychotic/neurotic, unipolar/bipolar, etc.

Among others, there are chapters on the seasonality of depression, seasonal variations in mania, and prospective studies in infraradian mood rhythms and SAD. There are contributions from Switzerland, New York and the National Institute of Mental Health (NIMH). Much of the book is devoted to phototherapy and its affect on melatonin, serotonin and noradrenalin.

Although this book martials an impressive body of evidence for the existence of SAD, this is mainly anecdotal and is mostly confined to the USA. There is as yet no large-scale study comparing SAD with non-SAD depressives. Since SAD is so dependent on the physical environment, questions arise concerning its correlation with latitude. It is surprising therefore to read (p. 119) that as yet only one study – that of Potkin *et al* – has on the basis of a newspaper advertisement, attempted to address this problem. The point is made that it would be worthwhile to design a more scientific study to assess the seasonal incidence of symptoms in different parts of the world – advice which should be followed by some of the investigators. A group of workers at the NIMH found that overnight production of melatonin and prolactin was reduced in 15 SAD patients. Other researchers, however, find that these changes are not confined to SAD patients.

Is all this anything new? A seasonal incidence of psychiatric disorders has been recognised since Kraepelin. Why then is it only now that SAD is described? If the incidence of SAD is maximal at latitude 40°N (i.e. approximately Washington, New York and California) why is it not more often described in Europe? What happens in the Southern hemisphere – Australia, South Africa and South America? If SAD is to do with bad weather conditions or lack of sunlight, why is it not more often found in the British Isles and Scandinavia – not to mention in the perpetual darkness of north Norway, Spitzbergen or on polar expeditions. Why were there then no cases during the British Antarctic expedition? In a sizeable proportion of the British SAD patients described, depression was not so much seasonal as related to bad weather and cloud cover.

This is not to knock gratuitously the concept. Some contributions to this volume were particularly interesting and informative. In a series of elegant experiments Checkley concluded that the antidepressant affects of bright light are more than a placebo. Thompson, comparing three different forms of light, concluded that augmented bright light in the middle of the day is the most efficacious.

Obviously a formal placebo-controlled double-blind trial is not feasible as light has to strike the retina to work. Indeed, we do not know how bright light is antidepressant. The suggestion that phototherapy is as effective as more traditional therapies is pure conjecture.

M. W. P. CARNEY, *Consultant Psychiatrist, Northwick Park Hospital, Harrow*

Exiles from Eden: Psychotherapy from an Evolutionary Perspective. By KALMAN GLANTZ and JOHN K. PEARCE. London: W. W. Norton. 1989. 319 pp. £25.00.

This book describes a form of psychotherapy in which the premise is that basic human attitudes and behaviours evolved in the context of primate hunter-gatherer bands. Human beings are therefore essentially adapted for this lifestyle and not having (yet?) evolved further to be adapted to agricultural society, let alone a modern industrial one, they will obviously have problems with the modern world. This mismatch is regarded as the basis of many mental health problems.

The hunter-gatherer groups are referred to as "the natural environment" (the 'Eden' of the title) and a number of attitudes adapted to this environment are described in the first part of the book. For example, the authors are in agreement with the views of others that characteristics such as reciprocity are inherited, having been selected by the reproductive success of groups whose members share. Child development, gender issues, male and female roles in society are similarly explored, and working hypotheses for psychopathology are described and derived from observation of the malfunctioning of these systems in the modern world.

The second part of the book is devoted to more clinical descriptions showing, for example, how lack of 'dominance' (equivalent to 'self-esteem' and differentiated from 'domineering') results in reactive problems of aggression. The authors work in a broadly cognitive-behavioural framework and concepts of which behaviours were adaptive in hunter-gatherer bands are used to guide and advise clients about the required behavioural and attitudinal changes that are needed for a happier life nowadays. Although it is clear from the case histories that there is regard for the importance of empathy and the therapeutic relationship, the importance of these aspects are not satisfactorily accounted for, and the question of dependency and problems of weaning from the therapist needs some elaboration. The clinical examples are vivid and focus as might be expected on vignettes which illustrate the theory in practice. The fact that much has passed between therapist and client before this is only referred to briefly, leaving this to the imagination in what are essential areas of work. In a short volume, designed to argue a particular point, this is probably not unreasonable, although I hope the authors will write further on this in future, in particular how they manage the defences and countertransference problems in the months or years before the client begins to make use of the framework they offer.

I found this an enjoyable book to read. The authors demonstrate that they are willing to work patiently with difficult clients over many years, and I found that the book added to my understanding of human functioning in social groups. For both of these reasons I would recommend this book to a wide audience.

TOM MURPHY, *Consultant Psychotherapist, Department of Psychotherapy, Royal Edinburgh Hospital, Edinburgh*

Conversations with Pre-school Children. Uncovering Developmental Patterns. By PAUL V. TRAD. New Haven/London: Yale University Press. 1989. 227 pp. £14.95.

The intention of this book is to acquaint health care professionals with the developmental process in pre-school children, in order that they may distinguish normal from abnormal behaviour. The author's basic tenet is that behaviour suggestive of psychopathology may actually be normal adaptive responses of young children encountering traumatic events. He contrasts the medical model, which he considers limited, with the new field of developmental psychopathology, which draws on knowledge from various scientific disciplines.

The book consists of a brief examination of the case histories of six pre-school children whose behaviour has caused concern. The clinical descriptions are accompanied by limited reports of conversations between the author and the child, mainly in question and answer form. Following each vignette is a lengthy discussion of theories of normal development pertinent to each case. A wide range of models and theories are used – primarily attachment theory, theories of temperament and also of cognitive development Research on children's behaviour, especially recent research, is quoted extensively, and the chapter on play behaviour is particularly comprehensive and erudite However, it is surprising that the pioneers of play therapy and psychoanalysis with children, Melanie Klein and Anne Freud, are not mentioned.

The children's behaviour is examined under various headings – cognition, locus of control, play behaviour, pro-social behaviour and aggression. It can be deduced from these titles that this is a very American book, which focuses on a very American preoccupation – that every child showing any disturbance of behaviour will automatically receive a diagnostic label under DSM-III-R. Even a description of normal sibling rivalry is discussed as possibly classifiable as the disorder 'phase of life problem'.

For my taste Trad focuses too much on the theory and insufficiently on the personal details of each case, with scant exploration of the environment of each child This lends a mechanistic impersonal feel to the book, with too rigid a consideration of psychopathology and a disappointing reluctance to give meaning to the children's behaviour. Finally, I feel the book falls short of its aim to provide a practical guide for professionals in this field.

STEPHEN ISAACS, *Consultant Child Psychiatrist, Waltham Forest Child & Family Consultation Service*

Suicide in Children and Adolescents. Edited by GEORGE MACLEAN, with contributions from S. DAVIDSON, R. T. JOFFE, D. R. OFFORD and C. R. PFEFFER. Ottawa, Ontario: Hogrefe & Huber. 1990. 144 pp. Canadian $26.00.

This slim Canadian volume covers a topic which is of interest to both child and adolescent psychiatrists and adult psychiatrists. Its five chapters cover subjects ranging from epidemiology and clinical assessment to the role of depressive disorders and risk factors in management of suicidal children and adolescents An increase in completed suicide in adolescents and young adults has caused concern both in North America and in the United Kingdom. The reasons for this are uncertain. Throughout this volume it is clear that research on suicidal attempts and completed suicide meets a gap in knowledge which is not yet filled by accurate research. The North American experience of the use of firearms by adolescents is not one seen in the United Kingdom. However, in other respects, the risk factors, outlined somewhat repetitiously, show that we need to be concerned about the mental health of children and adolescents who at the end of a road of disturbance may either attempt or complete suicide. This book is a helpful contribution to the field, but reveals once again the need for more research particularly into younger children with suicidal ideas. The role of depressive disorders in children and adolescents is stressed, something which in the past has been given a low priority in thinking about suicidal children.

In general this is an excellent book with a good review of the area, although more careful editing perhaps would have saved some repetition. The best chapters were those by Pfeffer on the manifestation of risk factors and that on management by Davidson. The ending of each chapter with a list of references with comments by chapter authors is a practice to be encouraged. This book is to be recommended to trainees in child and adolescent psychiatry and adult psychiatrists on call for general hospitals. However, since it is Canadian I am not sure whether it will be easily available and whether, in fact, British alternatives such as Keith Hawton's books would be more accessible.

L. G. SCARTH, *Consultant Psychiatrist, Department of Child & Family Psychiatry, Royal Hospital for Sick Children, Edinburgh*

Clinical Interviews with Children and Adolescents. By PHILIP BARKER. London: W. W. Norton. 1990. 153 pp. £14.95.

Many clinicians contemplating an interview with a child or an adolescent become panic-stricken and their usual competent interviewing skills evaporate. This can lead to doctors and others actually neglecting to interview children and consequently missing the diagnosis. I therefore approached this slim volume with some eagerness, hoping to find just the text to introduce trainees to the skills of interviewing children. The idea is an original one and in many ways this book admirably meets the needs of trainees and others by simplifying the process and offering the distillation of the experience of a sympathetic and skilled clinician. There are sections on interviewing young children, older children and adolescents, children with special problems of communication such as those who are mentally handicapped, deaf, autistic or psychotic, and a chapter on assessing the suicidal youngster. The structure of interviews including the proper way to terminate contact is given due attention, and this will be of help to novices who find termination the most difficult part of psychotherapy or counselling sessions.

However, in the end I found the book a disappointment. The author seems never to have got clear in his mind what his aims are. He seems to be considering therapeutic interviews as well as diagnostic ones originally and yet he misses them off the list of reasons for interviewing children! On page 12 he states that interviews "that impose change are used rather rarely", betraying his intention not to consider the interviews that aim for change (i.e. therapy or counselling ones). Yet in the chapter on termination, the author is clear that children become attached to those who are treating them and is advising accordingly. Some of the information is misleading. On page 127 he states: "suicide and suicidal behaviour are common in adolescents". It is true that clinicians commonly see such youngsters, but the behaviour is rare in adolescents, the majority of them never evincing such pathology. Those of us who have training responsibilities will find the chapter on interviewing children in most standard textbooks covers most of what we need and the rest must be supplied by example and carefully supervised experience.

DORA BLACK, *Consultant Child & Adolescent Psychiatrist, Department of Child Psychiatry, Royal Free Hospital, London*

Time-limited Intermittent Therapy with Children and Families. By THOMAS KREILKAMP. New York: Brunner/Mazel. 1989. 243 pp. $41.00.

Health Maintenance Organisations have developed rapidly in the United States in the last decade. They provide a type of medical care which is intermediate between general practice and routine National Health Service out-patient care, at a relatively low cost compared with the health care provided by individual medical specialists in their private offices or at major medical centres. Family medicine, equivalent to British general practice, is available in some areas, but most parents regularly consult a specialist paediatrician about their children, not only for serious illness but also for developmental checks, minor complaints, vaccinations, and advice about feeding, sleeping etc. for infants. These functions are more normally dealt with by health visitors and clinical medical officers in child health clinics in Britain. Each local centre for a Health Maintenance Organisation usually serves a population of between 10 000 and 60 000 people and therefore can provide specialist care on an out-patient basis by employing paediatricians and other practitioners including psychiatrists, psychologists, nurses and social workers.

Kreilkamp describes the therapy provided in the small-child mental health service of a Health Maintenance Organisation in Cambridge, Massachussets and contrasts it with the much more time-consuming approaches of traditional child psychiatric departments and private practice where individual psychoanalytical therapy and extensive family therapy predominate. The model he uses may be helpful for American mental health workers who are increasingly required by resource constraints to see large numbers of referrals of relatively psychologically healthy and motivated families. It offers relatively little to typical child mental health clinics in Britain where such families form a minority of referrals. The author admits that the system does not serve families well who are ambivalent about treatment, would not attend reliably, have multiple problems or are abusive.

In contrast to the economical form of treatment, the author's personal style is rather voluble. The meat of the book is contained in the second half which comes alive with clinical examples. He describes an active short-term therapy for children and families referred by paediatricians which draws on behaviour therapy, family dynamics and child development, and he frequently uses a consultation model with parents. He offers a few interviews in most cases, but does make direct contact with families over the telephone. Treatment for these parents whom he describes frequently 'hot-housing' their children, focuses on changing behaviour, limit setting and containing rather than expressing feelings. The aim is to "help them get on with life rather than to understand themselves perfectly". Some technical tips, particularly for adolescent problems, may be helpful to experienced general practitioners and child mental health professionals, but this type of approach should be, and already is, used only selectively in Britain because of the greater morbidity of specialist referrals.

ALYSON HALL, *Consultant Child and Adolescent Psychiatrist, The London Hospital and the Emanuel Miller Centre for Families and Children*

Facing Shame: Families in Recovery. By MERLE A. FOSSUM and MARILYN J. MASON. London: W. W. Norton. 1989. 192 pp. $9.95.

Fossum & Mason introduce their book by describing shame, an illusive and until recently largely neglected affect, as a dragon; a mythic monster. This description is valuable because it links the negative *and* positive powers associated with dragons to the often intangible feelings associated with shame. They say shame and dragons are both creatures with "... claws (which) can lock us in a frozen state and devour our ability to verbalise" (p. ix). Yet shame's invisibility can also provoke intense curiosity and a need to know more about oneself and one's family experience; thus possibilities of transformation or metamorphosis (a dragonish capacity) arise.

To develop their argument, the authors draw extensively from their clinical experience with individuals, couples and families. They exemplify the incapacitating nature of shame, and the processes by which it can open pathways to an individual's innermost self; this hopeful aspect no doubt arises *because* the two aspects of shame are noted, and because the authors have developed some feeling for the struggles their patients must undergo when trying to integrate the two. In addition, the authors explore the realm of family relationships where shame is illustrated as both a powerful inhibitor and a potential facilitator of genuine contact between individuals.

This is by its design a fairly wide-ranging book. However, three of the points which are made seem particularly worthwhile.

The first point relates to the origins of shame in families and the transgenerational nature which this affect possesses. That is, shame tends to be passed down the generations in families, which contributes to its illusiveness. Family systems are then inclined to sustain or 'inflict' the experience of shame in powerful and/or unconscious ways.

The second point is associated with the common tendency towards defensive cycles which maintain shame and inhibit relationships. Fossum & Mason represent these diagrammatically, which illustrates the unhelpful interplay between shame and the need to control oneself. These cycles are linked with addictive processes, and this section of the book would be of particular interest to those working with patients for whom addictions are a real problem.

The third point relates to the need for therapists to encounter their own shame in order to facilitate an openness to shame issues, and to the unconscious communications employed by patients and families in this area of experience.

This book is a worthwhile contribution, and would be of particular value to those readers who want an accessible introduction to the multifaceted aspects of shame.

PETER FULLERTON, *Analytical Psychologist, Melbourne, Australia*

Learning and Education: Psychoanalytic Perspectives. Edited by KAY FIELD, BERTRAM J. COHLER and GLORYE WOOL. Madison, USA: International Universities Press. 1990. 1016 pp. $65.00.

This volume comes from the Chicago Institute for Psychoanalysis and consists of 31 papers organised into four sections, each with an introduction reviewing the literature and linking the themes of the chapters. The quality of the papers varies, and it is a book to be dipped into rather than read at one go.

The first section surveys the major theoretical perspectives within psychoanalysis and developmental psychology as related to education. Piaget is frequently quoted, but the major emphasis is on issues of the self of the student and the interaction between self-esteem and learning; Kohut is the main influence. There is a shift away from the earlier idea that psychoanalytic theory could be used directly by school teachers and towards the study of what fosters the student's capacity to use the teacher's help. Muslin & Val's paper on the supervision of psychiatric residents focuses on this issue, with implications for all teaching situations.

Part 2 explores concepts of learning throughout life. I enjoyed three papers: Stott's "Making meaning together", which describes a five-year-old's use of writing within the family setting; Garber's "The child's mourning", which suggests that in a bereaved family a child's mourning may be blocked if its phases are out of time with that of the parent; and Stiver's "Examination of work inhibitions in women: a special problem for the female teacher and student".

The third section considers clinical applications, and includes papers on learning difficulties in adolescents with borderline disorder, special education, and the psychotherapy of infants and toddlers who are developmentally delayed.

The best part of this book is the last, most coherent, section: "Learning and the teacher". All the contributors are Institute staff members involved in its teacher education programme. The chapters reflect a collaborative attempt "to incorporate psychoanalytic knowledge of personality development and relationship dynamics and of the process of change into psychologically enriched programmes of teacher education". The final chapter by Field is totally engrossing. It gives in full the process notes from a 22-session course for classroom teachers run on the lines of Balint groups by an analytically trained instructor, and the author's comments on the process as it unfolds. This chapter illustrates beautifully the overall themes of the volume.

NICOLA BLANDFORD, *Department of Psychotherapy, Cambridge*

Managing Anxiety: A Training Manual. By HELEN KENNERLY. Oxford: Oxford University Press. 1990. 177 pp. £10.95.

This book offers a clearly articulated and constructed summary of many of the current practices of cognitive behaviour therapy for anxiety. It is written for health care workers in the primary care setting although some psychologists and psychiatrists will also find it useful. It is not to be read as a book but rather, as I believe the author intended, as a step-by-step approach to intervention. Kennerley takes us through the process of graded tasks and exposure to feared situations, eliciting a hierarchy of fears, eliciting dysfunctional thoughts and challenging these thoughts, homework, dealing with avoidance, controlling symptoms (e.g. breathing control), enhancing self-control, and ending therapy. The text is mixed with short case presentations (although I am unsure what to make of the authors claim that: "Any similarity with real persons in the case histories is coincidental.") Kennerley also provides helpful work sheets that maintain the step-by-step approach. There is also advice on relaxation and how to make ones own relaxation tapes, on tranquilliser withdrawal and sleep problems.

The writing is very to the point and would need to be supplemented with wider reading on the nature of anxiety disorders, their relation to depression and other psychiatric conditions. There is also rather little recognition of the importance of personality disorder which is becoming identified as one of the main reasons that treatments do not always go as the manuals suggest. More discussion of the fact that relaxation is not always helpful is also needed, and also the limits of the approach. The role of the therapeutic relationship is mentioned almost in passing. At times the print goes into black on grey format which I found irritating to read and could see no useful reason for.

This kind of approach will not be everyone's cup of tea. Still, taken on its own terms it is as good as many and better than most. It is a highly structured, well presented and confident account of one approach to anxiety and one that its target audience may gain much from.

PAUL GILBERT, *Principal Clinical Psychologist, Pastures Hospital, Derby*

Stress Management: An Integrated Approach to Therapy. Psychosocial Stress Series No. 17. By DOROTHY H. G. COTTON. New York: Brunner/Mazel. 1990. 288 pp. $30.00.

This is a comprehensive account of stress and its management which is well written and readable. The initial chapter, an overview, introduces various concepts of stress and gives a general plan of the book which seems logical and clear. The following three chapters deal with the definition of stress, its association and relationship to illness, and methods of assessment. A good review of the recent knowledge, research and theories on the subject is provided.

Then follows a chapter dealing with the planning of treatment for each individual patient/client. This gives a particularly useful method of drawing together all the data collected and producing an individualised treatment plan; it uses a pictorial grid which clarifies the components of therapy and how they interact. It emphasises that the common belief that everyone with stress requires the same form of therapy, usually consisting of the nebulous 'relaxation training', is mistaken. As the title of the book implies, an integrated approach to therapy is suggested.

The remaining chapters give a comprehensive account of the various forms of stress management, divided into the physiological (i.e. relaxation therapies and life-style management), cognitive, and finally behavioural approaches. The theoretical perspective and developmental history of the treatments are described and also a general description of the process of treatment is provided. Clearly, to learn more about the individual forms of treatment one would need to read elsewhere, but adequate references for further reading are given.

Throughout the book, three case vignettes are used to link the chapters together. Although this initially seems rather contrived it does provide useful examples of how to assess and plan treatment. These cases also show how very different clinical situations can be approached in a uniform way. This is a well balanced book which is clearly directed towards an American market but can also be adequately utilised by the British professional dealing with stressed individuals.

R. A. WILLIAMS, *Psychiatric Registrar, St James' University Hospital, Leeds*

Stress and Medical Procedures. Edited by MARIE JOHNSTONE and LOUISE WALLACE. Oxford: Oxford University Press. 1990. 184 pp. £25.00.

The recently published government report on pain exposed the shortcomings of the medical profession's ability to control pain and manage patients with post-operative pain. Such documents draw attention to the fact that modern medicine, for all its high-tech excellence, has a psychosocial dimension that should not be ignored. Hence the need for this timely book, which is divided into two sections. The first includes a theoretical review of stress and the second outlines stress in various medical settings. Included in the latter are diagnostic procedures, medical and surgical treatments, and obstetric and paediatric procedures.

The scene is set by the co-editors who contribute a useful preface and concluding chapter. They point out that the complex interaction of variables that have to be measured in research of this nature include not only features of the stressful stimulus (intensity, duration, etc.), but also the individual's appraisal and expectations (perceived control, social supports, etc.) as well as the diversity of responses.

Steptoe contributes an excellent review of the factors that influence the pattern of psychobiological responses during stress, and describes an interesting study of subjects who were asked to attend to and analyse the sensations experienced during a disagreeable procedure. Far from increasing physiological reactivity (as might be expected), the responses in these subjects were attenuated compared with those who had received no special instructions. This kind of research finding could be translated into clinical practice.

One theme running through this book is that the coping style of each individual subject is a critical determinant of the outcome of a medical procedure. This means that the therapist has to utilise existing patterns of behaviour to help each patient cope more effectively with the stress invoked by the particular procedure. Because some patients display an active, information-seeking mode of response they would be better suited to an approach in which specific information of a procedural and sensory nature was provided. Whereas those who cope with stress through avoidance means and depend on the health care provider for support would perhaps be better served by provision of information of a more general, less descriptive, and less threatening nature.

The authors of the chapter on paediatric procedures draw attention to the potential cost-effectiveness of procedures that might reduce time in hospital, need for analgesia, psychiatric morbidity and other consequences of painful hospital procedures. As we move into the 1990s and hospitals contract out their medical and surgical services to purchasers of health care, health psychologists have an extra incentive to develop this field of enquiry and to translate the findings of their research into practical (and financial) realities. This well edited book provides a useful database and I recommend it for those wishing to investigate this increasingly complex field.

CHRISTOPHER BASS, *Senior Lecturer in Psychological Medicine, King's College Hospital, London*

The Biological Basis of Psychiatric Treatment. Edited by S. GERSHON and R. POHL. Basle: Karger. 1990. £118.40.

This volume is the third of a series called "Progress in basic and clinical pharmacology". It is a multi-authored volume and covers not only treatment aspects of the most common and important psychiatric conditions, but also the available evidence for a biological aetiology of schizophrenia, depression and anxiety disorders The main strength of this book is an attractive philosophy of mixing information about complex neurotransmitter physiology with clinical aspects of the common psychiatric disorders and their management. This makes it more readable than most texts on this subject.

Most chapters outline the historical background for the biological approach to the conditions described, and detail both animal and human studies with an appropriate emphasis upon the limitations of the former when applied to psychiatric conditions in the latter. The biological rationale for the physical treatments used in psychiatry is placed in perspective with other treatment modalities known to be effective. The evidence for links between psychological theories of illness and central neurotransmitters is reviewed.

I have some reservations about this volume. In particular, this field is a fast moving one, and by the time such an edition is found on the shelf, there are, inevitably, a number of areas requiring updating. In addition, the cost makes this volume more attractive to libraries that to individuals. It is, however, recommended as a thorough and readable reference source for the biological basis of psychiatric treatment.

R. E. B. WHITE, *Lecturer in Psychiatry, Department of Mental Health Sciences, St George's Hospital, London*

Body Self and Psychological Self: A Developmental and Clinical Integration of Disorders of the Self. By DAVID W. KREUGER. New York: Brunner/Mazel. 1989. 180 pp. $34.00.

Focusing on the treatment of different cases of narcissistic pathology such as addictions, eating disorders, psychosomatic symptoms, etc., the author offers a condensation of recent theoretical ideas about the development of the self. He tries to show how these ideas can be integrated into clinical practice, with many examples given. He relies mainly on the fairly recent theory of the late Heinz Kohut, a well known psychoanalyst in Chicago. Kreuger tries to blend these newer theoretical concepts into a sequence of theory from Piaget onwards. A lot of work has gone into this book, and I am sorry to say that I do not think Kreuger has succeeded in his aims.

The meaning of the term selfobject, written as one word, is not made clear. It is the hallmark of the Kohut School, which has raised considerable controversy in America and has not been highly regarded in the UK. For those who know enough about psychoanalytical theory to recognise the terms used, this book is too elementary and does not tackle the status of Kohut's ideas. For those who do not know much theory, there is not enough explanation of the terms nor of the psychoanalytical contexts in which they exist. It would be quite easy to imagine from the text that the ideas put forward are all of an equal status and attract equal regard in the world of psychoanalytical theory, which is far from the case.

In addition there is a hidden and deep-seated controversy in this book, which revolves around the idea, first put forward by Ferenczi, of what came to be called 'the corrective emotional experience'. The argument is essentially about what causes the changes in psychoanalytical treatment. How does the talking cure work? There are two main runners: the undoing of unconscious conflict by the demonstration of the unconscious historical content of present day ideas and feelings so that the analysand can compare in his mind past and present reality; or the undoing of unconscious conflict by the new experience of the behaviour of the analyst in spite of the emergence of destructive fantasies The problem with the second one, the corrective emotional experience, is that it lends itself to abuse by the therapist, it invites short-cuts, and it has never really caught on as the answer to how psychoanalysis works. The psychoanalytic community worldwide has preferred the interpretation of the transference as the therapeutic factor. Although not explicitly stated, this book relies on the idea of the corrective emotional experience as the curative factor, rather than the interpretation of the transference.

This book shows the difficulties that Kohut's followers are experiencing in trying to integrate their theoretical stance into mainstream psychoanalytical thinking.

MICHAEL R. POKORNY, *Psychoanalyst and Psychotherapist, London*

Productive and Unproductive Depression: Success or Failure of a Vital Process. By EMMY GUT. London: Routledge. 1989. 275 pp. £30.00 (hb), £12.95 (pb).

Much of the time spent by psychiatrists, psychologists and others is occupied with aspects of depressive illness, and in particular with those 'difficult' cases in which usual treatment strategies seem not to work. We are all aware that the genesis and maintenance of depressive states depends on a complex interplay of biological, psychological and social forces which so often seem to combine to lock our patients into prolonged, intractable periods of low mood, anhedonia, introspection and lethargy. Trying to make sense of these forces, and to relate them to the suffering individuals we strive to help, can seem an impossible task.

Gut addresses this task with considerable fortitude, and not a little originality. Drawing on her own clinical experience, but also on the writings of many other authors, she proposes a theory that there is a 'basic depressed response' as a discrete mood state similar to the emotions of anger, fear, joy, shame and others. It is worth mentioning at this point that although Gut writes firmly from the tradition of dynamic psychotherapy her language is clear and mercifully jargon-free, all terms are defined if they are to be used in a technical sense and her clinical material is firmly rooted in the everyday world of clients that everyone will recognise.

The basic depressed response is seen as a genetically based mechanism for helping us to cope with specific problems, it is (in Gut's own terms) "a potentially adaptive response (evoked) whenever we perceive, largely unconsciously, that a significant physical or psychological effort of ours is failing in its purpose ... and when we cannot grasp what is amiss". The depressed response is thus equivalent to a state such as grief or anger that can help us deal with problems, but can equally become maladaptive as coping mechanisms.

This book reviews the nature of the primary depressed response, compares it with and differentiates it from a number of other mood states (including depressive illnesses in the DSM–III–R or ICD–9 sense) and discusses the ways in which the adaptive function can fail or be blocked – 'unproductive depression' – leading potentially to severe or prolonged states of depression. There are useful chapters on family and sociocultural influences on depression. The real strength of the book, and what brings it to life, lies in the richness of the illustrative material, which is taken from Gut's own clinical work, published work of others, experiences of friends and colleagues and, most strikingly, an extended discussion of depression as depicted in various members of a fictional family in a novel by Chaim Potok.

Although owing a great deal to other authors (a debt freely acknowledged, not least in the dedication to John Bowlby who himself provides a foreword), this is an original and exciting book. I would recommend it to all psychiatrists, and any others interested in understanding depression, as an accessible, thought-provoking account of a different way of conceptualising an all too familiar problem for us all.

GRAEME MCGRATH, *Consultant Psychiatrist, Manchester Royal Infirmary*

Self-Disclosure in the Therapeutic Relationship. Edited by GEORGE STRICKER and MARTIN FISHER. New York: Plenum Press. 1990. 295 pp. $45.00.

The relationship between psychotherapist and patient has been a controversial and much-debated issue ever since Freud suggested that the analyst should be "opaque to his patients, like a mirror, and show them nothing but what is shown to him". The problem started right there, for while Freud was recommending this ascetic stance to "physicians practicing psychoanalysis" we know that he frequently did not follow his own rules. Is the therapist's 'self-disclosure' always undesirable although often unavoidable? Or is the total denial of it not only impossible but even untherapeutic? In this collection of 18 papers by American practitioners these questions are discussed at some length and with admirable lucidity.

The spectrum of the discussion is wide. It considers its problem in the framework of classical analysis, group therapy and feminist therapy. It includes work with children and adolescents, non-white ethnic minorities and holocaust survivors. Within the limits of this review, it is of course impossible to refer to the many controversial issues which these papers throw up.

Summarily it can be said that therapists who stress the undesirability of self-disclosure adhere to the psychoanalytic assumption that therapeutic success is rooted in the development of a transference which depends on the neutrality of the therapist On the other hand, therapists advocating self-disclosure stress the importance of a warm and open relationship between therapist and patient.

In the excellent chapter on "Feminist therapy perspectives" (Brown & Walker) the discussion of this issue is particularly differentiated: although from a feminist point of view the unequal distribution of power between a silent impersonal therapist and a self-revealing patient is of course unacceptable, self-disclosure of the therapist is itself open to abuse and can oppress the patient.

This feminist analysis invites us to have a closer look at the meaning of self-disclosure The assumption seems to be that we disclose ourselves by talking about ourselves. But do we not often hide behind words? And do we not often reveal ourselves through silence? A more heretical question: what is it we disclose when we disclose our 'self'? We seem to make the assumption that there is a well-defined self, independent of situation and context, to be disclosed or kept hidden.

Talking about self-disclosure in the way in which most authors of this collection do is like talking about a technique, a strategic move, to be used or not used In my view, the quality of therapy depends on the quality of what happens *between* the therapist and the patient. The effect of strategic moves on the quality of this in-between is, I think, more limited than we choose to admit.

HANS COHN, *Psychotherapist, Richmond, Surrey*

Microcomputers, Psychology and Medicine. Edited by ROBERT WEST, MARGARET CHRISTIE and JOHN WEINMAN. London: Wiley. 1990. 320 pp. £39 00

This is a wonderful book with a misleading title. It is in fact a guide to the use of computers in psychological aspects of medicine, rather than in psychology *and* medicine The contributors seem to have been asked to assess the impact of microcomputers in one field of psychological medicine, and this they generally do through considering their own work. Although an excellent idea, these illustrative examples do not always ensure a balanced view of the psychological literature – but, in fairness, they never claim to do so. Considerable effort has been made by all contributors to ensure that basic principles (rather than specific applications or technology) are emphasised: the book will not become obsolete as soon as the IBM-PC or BBC micro finally fall from grace.

The 16 chapters (grouped loosely into six sections) are generally of a very high standard, and are so free from computing jargon that they can be read by the complete novice. Most are concerned with psychological assessment in some shape or form, covering topics as diverse as animal behaviour, reaction times, clinical anxiety and neurological assessment as well as the inevitable computerised questionnaires. Nearly all of the chapters describe in some detail the advantages and disadvantages of using microcomputers for these applications, and several give useful details of the cognitive test batteries which have been assembled for specific applications together with a precis of the results from the research projects. Other clinically relevant chapters cover the role of microcomputers in therapy and training of psychiatric and aphasic patients, and applications for those with special needs.

There are in addition an exceptionally clear couple of chapters dealing with data analysis by microcomputer and the development of microcomputer applications. The store of wisdom in this last chapter alone justifies the price of the book. It contains sound practical advice of the kind that can normally only be obtained through bitter experience, and it is quite possible that someone who is contemplating setting up a research project involving microcomputers could save themselves months of wasted effort and a mountain of useless hardware by reading this chapter.

Given the enormous potential of microcomputer applications in psychological research this book will probably have a much broader appeal than its publishers seem to anticipate. Although seemingly aimed at clinical researchers, my copy has been enthusiastically read by undergraduate and postgraduate psychology students, clinical and occupational psychologists, and biomedical researchers. I hope for my students' sake that a more reasonably priced paperback edition may soon emerge.

COLIN COOPER, *Lecturer, Department of Psychology, University of Ulster*

Trauma and the Vietnam War Generation: Report of Findings from the National Vietnam Veterans Readjustment Study. By RICHARD A. KULKA *et al.* New York: Brunner/Mazel. 1990. 352 pp. $19.95.

In 1980 the American Psychiatric Association adopted post-traumatic stress disorder (PTSD) as the up-to-date label for mental disturbance caused by trauma. Observant carers have known about this problem for much longer than a decade, of course. One interesting thing about the 1980s has been the definitive extension of the concept into situations non-military; although military machinations probably generate the majority of PTSD. Unfortunately, the military has not always been adept at acknowledging this particular psychopathology. Lessons misremembered need to be relearned. The book aims to educate about the mental aftershock of armed conflict. It is the 'friendly' version of the National Vietnam Veterans Readjustment Study (NVVRS).

When 'Vet Centers' were set up by the Carter-established Readjustment Counselling Service in the 1970s it was believed that they would do their job quickly and close. Instead, veterans kept coming, especially because of what was soon called PTSD. Many Congressmen were sceptical about a continuing need; others saw it clearly and insisted on it being met. Senator Alan Cranston was the man prepared to walk the walk and not just talk the talk and he ensured that the NVVRS was mandated by Congress in 1983 to establish the prevalence and incidence of PTSD and other psychological problems among returned veterans. Senator Cranston writes the foreword to this book of the study.

This is certainly one of the largest mental health epidemiological studies ever undertaken, and the authors express an amusing egotism in equating its importance (in the preface) with the endeavours of the early American astronauts. The amount of psychopathology unearthed is enormous; 486 200 veterans are current PTSD sufferers; 350 000 others retain lesser symptoms. There is considerable recommendation for further research. There is a final chapter by twice wounded ex-marine Grady, which contains good advice for sufferers and quotes from Henry IV part 1. Yes, Shakespeare knew about PTSD too.

The book aims to be widely read. It could be user-friendlier; certainly many will prefer a less complicated edition; but it reports a most important study well, and should be read by everyone interested in warfare and PTSD.

DOUGLAS S. C. GAMBLE, *Senior Specialist Psychiatrist, Queen Elizabeth Military Hospital, London*

Motherhood and Mental Illness: 2. Causes and Consequences. Edited by R. KUMAR and I. F. BROCKINGTON. Guildford: Butterworth Scientific. 1988. 294 pp. £42.50.

The first volume under this title appeared in 1982 and coincided with the formation of the Marcé Society, an international society dedicated to improving the understanding, prevention and treatment of psychiatric disorders related to childbearing. As well as evaluating what has changed in the intervening six years, this follow-up book serves as a progress report on the activities of the Marcé Society and hopes to be but the second in a series of volumes. The subtitle, "Causes and consequences", adequately covers the wide-ranging contributions included.

There are 11 chapters on relevant topics by authors from several disciplines, although, not surprisingly, psychiatrists are the most numerous. Subjects include: a comprehensive review of post-partum depression; sociocultural aspects; endocrine and biochemical studies and the relevance of interactions between hormones and the central nervous system (CNS); the developmemt of an integrated, community-orientated service for severe post-natal mental illness; and three chapters on aspects of loss – a review of categories, causes and consequences of infant death by Bluglass, the active management of perinatal death by Savage and the sibling's perspective of loss of a baby by Dyregrov.

As a child psychiatrist, I particularly valued those chapters concerned with the mother–infant relationship. Murray's fascinating research on early mother–infant interactions makes delightful reading. The very first chapter, on the nosology of puerperal mental illness by Brockington & Cox-Roper, is admirable for its clear exposition of a difficult area.

Most disappointing are those chapters on endocrine and biochemical studies and the relevance of hormone–CNS interactions to puerperal psychological changes, not for any want of erudition or lucidity, but simply because in spite of all the recent research effort there is still little positive evidence to support the hunch that 'hormones have something to do' with puerperal disorder. A similar sense of frustration is left by the very thorough review of non-psychotic post-partum depression by O'Hara & Zekoski.

There is something for almost everyone in this collection of papers. Each is well researched, up to date, and can, in fact, be read as a separate entity. There are the inevitable overlaps if one systematically reads the whole volume. Because few individuals would find the entire contents relevant to their work, the book would be most useful for departmental libraries.

PATRICIA AINSWORTH, *Lecturer in Child and Adolescent Psychiatry, University of Manchester*

Explorations with Families: Group Analysis and Family Therapy. By ROBIN SKYNNER, edited by JOHN R. SCHLAPOBERSKY. London: Tavistock/Routledge. 1990. 439 pp. £14.99.

This book is a collection of clinical papers and lectures by Skynner. The editor has organised these into three sections: (a) an introduction to the basic ideas and general principles in family therapy and group analysis; (b) special applications of family therapy and the group analytic approach (e.g. in general medicine or child psychiatry); and (c) papers illustrating Skynner's own approach, with an integration of ideas from group analysis and family therapy. This organisation, and the selection of papers, aims to chart the development of Skynner's clinical work and thinking and, in particular, his application of ideas from group analysis to family therapy. Topics vary from the general (e.g. marital problems) to the specific (e.g. school phobia) and some were written for particular audiences (e.g sexual counselling in general practice). The editor provides a useful introduction to each paper.

Taken as a whole, the book provides a good introduction to different approaches to symptomatic behaviour, and to how these fit together and complement each other. This is a welcome change from the more familiar stance of competition between different schools of thought, with each claiming primacy in the understanding and/or treatment of human problems Skynner is a great integrator, and the papers here illustrate his persistent willingness (indeed insistence) to move freely between different levels of explanation and theoretical models. His obvious curiosity (surely the most vital requirement for a clinician) shines through, and this is harnessed to an impressive rigour of thought and attention to detail.

Skynner also has that precious ability to make complicated ideas easily accessible, although this clarity is sometimes obtained at the price of some loss of complexity. However, even the sophisticated reader will be grateful for Skynner's articulation of what he knows but would find hard to say.

So far so good, but I do find a problem in who to recommend the book to. The papers vary a good deal, both in the topics covered and in their level of theoretical sophistication, and some are targeted at particular professional groups (e.g. physicians, marital therapists) I personally found the last section of the book the most interesting. Included here are papers about Skynner's own approach to family therapy, a way of working that combines psychoanalytic, group analytic and systems ideas. In particular, his 'use of self' in family therapy stands out as an important contribution to the field. The person of the therapist, and his motivation for this peculiar choice of work, is never far away from Skynner's attention and his openness to using himself to illustrate this aspect of therapy is an example to us all.

In conclusion there is something for everybody in this book, but the reader may also find some chapters of little interest. As the title implies, it will be of most interest to clinicians working with families. However, the integration Skynner achieves between different schools of thought, together with his philosophy of therapy, makes it a rewarding read for all clinicians who retain an open mind on their work.

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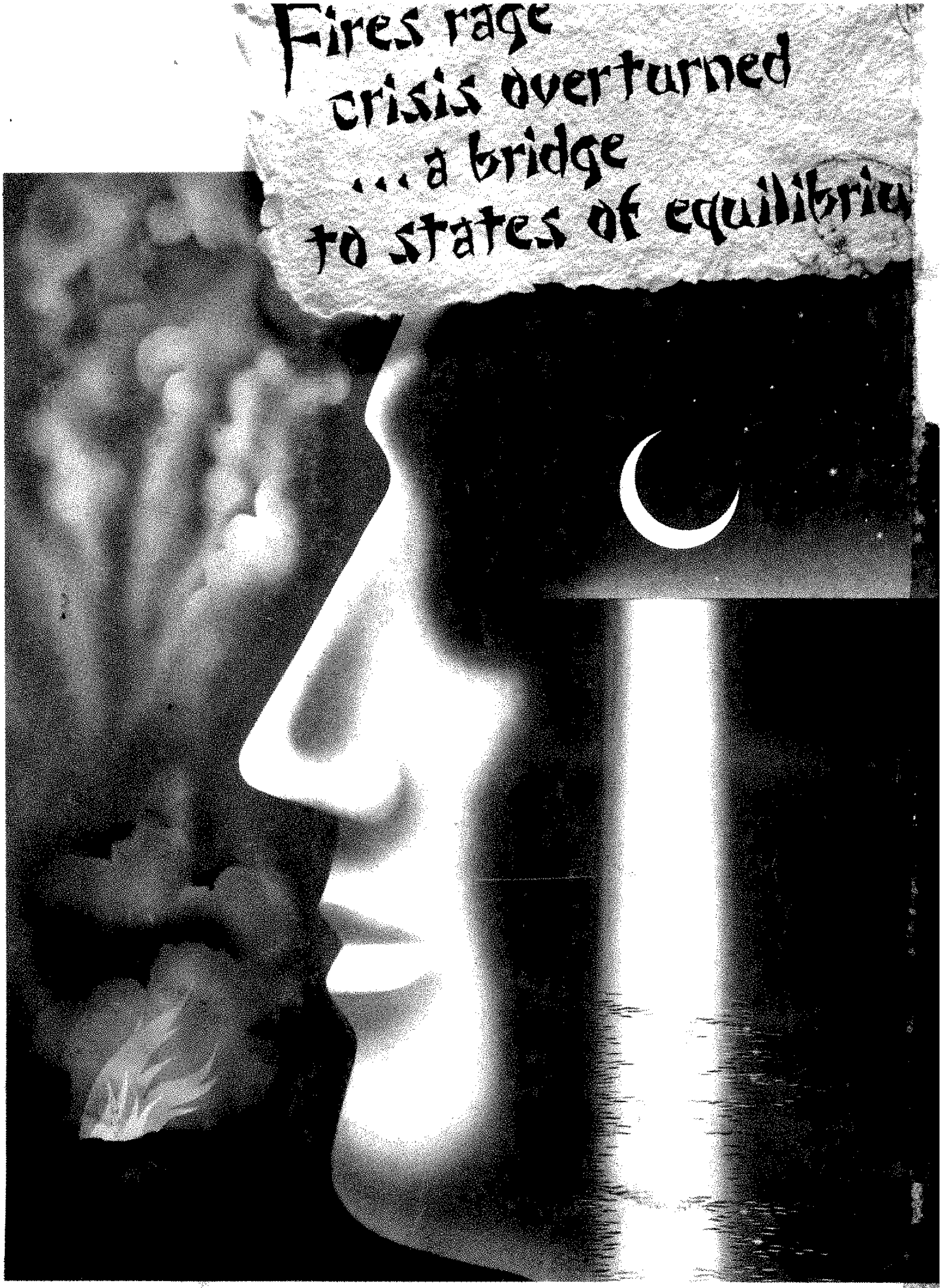

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